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A JOURNAL OF NEUROLOGY.

VOL. IX.

BRAIN:

A JOURNAL OF NEUROLOGY.

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APRIL, 1886.

Original Articles.

A CONTRIBUTION TO THE COMPARATIVE STUDY OF CONVULSIONS.

BY J. HUGHLINGS-JACKSON, M.D., F.R.C.P., F.R.S.

Physician to the London Hospital, and to the National Hospital for the Epileptic and Paralysed.

1. *Introductory.*

When I first began the investigation of nervous diseases I supposed, as most other physicians then did, and as perhaps most still do, the seat of epilepsy to be the medulla oblongata. But I soon came to the conclusion, that the cerebrum is the seat of epileptiform seizures (convulsion beginning unilaterally). I supposed the particular parts diseased to lie within the region of the middle cerebral artery; that rough localisation was before I had the advantage of the experimental researches of Hitzig and Ferrier. Later on, I supposed that, as I crudely put it, epilepsy (the epilepsy proper of authorities) depended on discharges of convolutions "at a greater distance from the motor tract" than epileptiform seizures did.[1] I believed then, that no variety of convulsion in man arose from any sort of change below the cerebrum proper. But very lately I have come back to the belief, that *some* convulsions (I do not call them epileptic) in children (and, I suspect some in adults which are *called* epileptic) depend on lesions of the pons or medulla oblongata. The fits in children to which I especially allude are "inward fits," otherwise called laryngismus stridulus; they are "respiratory convulsions."

---

[1] Study of Convulsions, 'St. And. Med. Graduates' Trans.,' vol. iii. 1870.

Of course I knew long ago that Brown-Séquard had demonstrated that in guinea-pigs a *liability to* convulsions could be experimentally produced (in ways everybody knows), and that the fits could be artificially brought on when the brain proper had been taken away. And I knew that Kussmaul and Tenner had produced convulsion by rapidly bleeding rabbits when the brain proper had been removed. It is plain that the convulsions in these animals do not depend on the cerebrum. Moreover there are the most valuable experiments of Westphal, another way of producing a liability to convulsions in guinea-pigs. But I supposed, erroneously I now think, that no sort of convulsion occurred *in man* from any kind of disease lower than the cerebrum proper. I thought the reason why convulsion occurred in certain lower animals deprived of the cerebrum, and not in man from disease of parts lower than the cerebrum, was explainable by the manifest great differences in the degree of evolution of the nervous systems of those animals from that of the nervous system of man. Seeing that the convulsions in the animals just mentioned were demonstrably owing to changes in the medulla oblongata, or pons, or both, I tried to show why such convulsions occurred in these lower animals and not in man.[1] Having now modified my belief on this matter, I come to attach even a greater value than I once did to the researches of Marshall Hall, Brown-Séquard, Kussmaul and Tenner, and Westphal. This, by way of mere preliminary. I now think, as above stated, that there are three classes of fits in man. I go on to speak more particularly of the three.

2. *The Scale of Fits.*

I believe (1) epileptic fits (epilepsy proper) to depend on "discharging lesions" of parts of the highest level of evolution, and (2) epileptiform seizures to depend on "discharging lesions" of parts of the middle level of evolution. Both (1 and 2) are cerebral convulsions. Both, to my thinking, are "cortical." (3) Inward fits (respiratory convulsions) and some other fits depend, I submit, on discharges beginning in parts of the lowest level of evolution.

---

[1] 'West Riding Asylum Reports,' vol. iii. p. 342. "On Evolution of Nervous Centres."

3. *The Three Levels of Evolution of the Central Nervous System.*

I now give a brief outline of the hierarchy of the nervous centres. It is admittedly imperfect; it is incomplete, too, by omission of the sympathetic chain, peripheral ganglia, and ganglia of the posterior roots of the spinal nerves. (Strictly, we ought to speak of four levels, the periphery being the real lowest.) It will be seen that the scheme is not after the morphological divisions, spinal cord, pons Varolii, &c., but is, especially, according to degree of indirectness with which centres represent parts of the body, and is thus an anatomico-physiological scheme. The motor nuclei of the pons Varolii and medulla oblongata are some lowest motor centres as much as the anterior spinal horns are some others.

(1) The lowest level of evolution, or series of lowest centres, is pretty much what Marshall Hall called "the True Spinal System," which, according to him, especially consisted of spinal cord, medulla oblongata, and pons Varolii. The nuclei for ocular muscles in the floor of the aqueduct of Sylvius are, no doubt, the topographically highest known lowest motor centres.[1]

The lowest sensori-motor centres represent all parts of the body, animal and organic, "from nose to feet," in simplest, &c., combinations.

Besides the centres we have spoken of, anterior and posterior spinal horns and their homologues higher up, and besides Clarke's visceral column and its lower and higher representatives, there are what are called "regulating centres," —micturition centre, vaso-motor centre, respiratory centre, &c. It may be that these are simply re-combinations of elements of the nuclei mentioned. I think that these centres are products of internal evolution.

---

[1] There is no impropriety in this expression, nor in such a one as "two lowest centres," since "lowest centre" is a proper name.

(2.) The middle level of evolution consists of Hitzig and Ferrier's motor centres, and of Schäfer and Victor Horsley's trunk centres, and also of Ferrier's sensory region.[1]

The middle sensori-motor centres re-represent all parts of the body, organic and animal, from nose to feet, in more complex, &c., combinations.

(3.) The highest level of evolution consists of parts of the brain in front of the middle motor centres (frontal or prefrontal, lobes), highest motor centres, and of parts behind the middle sensory centres (occipital lobes), highest sensory centres.

The highest centres re-re-represent all parts of the body, organic, and animal, "from nose to feet," in most complex, &c., combinations.

The lowest level of central evolution being evolved out of the periphery (epi- and ento-) represents all parts indirectly, but yet most nearly directly of all the levels; the middle, evolved out of the lowest, represents (re-represents) all parts doubly indirectly; the highest evolved out of the middle, represent (re-re-represent) all parts triply indirectly.

The highest centres, the climax of the evolution, are in popular language the "organ of the mind." I again insist, as I have done for many years, that this "organ" is as certainly of sensori-motor constitution as is the lumbar enlargement (some lowest centres). But the constitution of the highest centres differs vastly in degree from that of the lowest centres. They (*a*) represent most indirectly (re-re-represent) (*b*) literally all parts of the body having nerves (as the lower levels do), but they (*c*) represent all parts in most numerous and most complex, &c., combinations. Just as any state of consciousness is a state of a whole person psychical, so the correlative activity is of nervous arrangements representing a whole person physical (the whole organism). When a person is having a particular mental state, there are occurring physically, of course, activities of his highest centres. But what I urge is that the nervous arrangements then engaged represent his body, and some parts of it most especially.

---

[1] Since Hitzig and Ferrier are not agreed on details of motor representation by the cortex, it will be seen that by using the expression "Hitzig and Ferrier's motor centres," I do not pretend to demarcate exactly the middle and highest motor-centres.

I believe that the cerebellum also represents all parts of the body, but in ways different from the cerebrum. It will be seen that the lowest level of evolution is at once lowest cerebral and lowest cerebellar.

4. *The Comparative Study of Convulsions; Varieties of Lowest Level Fits.*

One object is to make a comparative study of the three classes of convulsions or fits depending on discharges on the three different levels of evolution,—lowest, middle and highest, levels of evolution. Speaking roughly, there are supposed to be lowest, middle and highest "level fits."[1] Several things qualifying a direct comparison and contrast of the three classes of fits will be spoken of later.

An "inward fit," or respiratory convulsion, is but one variety of fits on the lowest level, just as convulsion beginning in one hand is but one variety of epileptiform seizures (middle level fits), and just as convulsion ushered in by an "epigastric sensation" is but one variety of epilepsy proper (highest level fits). Moreover what we call respiratory convulsion should in many cases, since the convulsion becomes universal, be called respiratorily beginning convulsion. In each class of fits the convulsion is universal if the discharge be severe enough. Ordinary spasmodic asthma in the child or adult is a respiratory convulsion, and is also a lowest level fit. Both it and inward fits are, I submit, owing to sudden, &c., discharges beginning in some part of the respiratory centres. Rigor is another lowest level fit, dependent on discharge beginning in some part of the vasomotor centre. I suppose, as implied in some foregoing remarks, that the fits induced by Brown-Séquard and Westphal in guinea-pigs, and by Kussmaul and Tenner in rabbits, are lowest level fits in those animals. I shall consider later whether, as I have said I suspect, some fits in the adult called epileptic are really epileptic (epilepsy proper), or are lowest level fits—fits analogous to those occurring in Brown-Séquard's guinea-pigs. But I ought to say now, that I fear I shall not be able to make out anything like a demonstration on this matter. In any case we should have to consider the vast differences in the degree of evolution of the nervous centres in man and in the guinea-pig.[1]

---

[1] In still other words, which must not be taken literally, there are "least evolved," "higher evolved," and "most evolved" fits. Thus a severe epileptic seizure is a "more evolved fit" than a severe epileptiform seizure is, even when in each the convulsion is universal. I shall no longer use such expressions, for strictly the three classes of fits are three different dissolutions *being effected.*

It may be well to say very explicitly, what is, however, repeating part of the foregoing, that I do not believe epilepsy, the epilepsy proper of nosologists, and epileptiform seizures to be owing to any sort of lesion on the lowest level of evolution, although of course the motor centres on this lowest level are secondarily engaged in epileptiform seizures, and tertiarily in epileptic seizures.

"Inward fits," or respiratory convulsions, some lowest level fits, are the main subject of this paper; epileptic seizures, highest level fits, have been spoken of at the outset for the sake of order, and will be considered again in comparison and contrast with "inward fits," and other lowest level fits.

5. *Inward Fits.*

"Inward fits," or laryngismus stridulus (respiratory convulsions), occur mostly in children under one year of age, not often after two. Laryngismus stridulus occurs especially, Niemeyer says, during the first year of life; Charles West found that out of thirty-seven cases, thirty-one occurred between the age of 6 months and 2 years. Meigs and Pepper write:[2] "Of thirty cases selected indifferently from our practice, and from authors in which the age is given, thirteen were 6 months or less of age, eleven between 6 months and 1 year, four between 1 and 2 years of age, one of 2 and one of 4 years of age; so that of the thirty, twenty-four were under 1 year." "Laryngismus seldom attacks children more than a year old."[1] Henoch writes that it is "confined almost exclusively to the period between the 6th and 24th months. I have hardly ever observed it at a later age, but have seen cases as early as the fifth or sixth week."[2] What is the significance of this limitation by age?

---

[1] Since writing this sentence, I have seen fits in a boy whose case is, I think, very like the cases of Brown-Séquard's guinea-pigs. A boy of seven, falls suddenly to the ground when his head or face, either side, is touched; his face flushes, his eyes turn up and to the right, his respiration stops, and there is a sudden jerk of his limbs. Since his fits began a few months before the age of two and a half years (when he had "an ordinary epileptic fit"), as he has imperfect left hemiplegia, and for other reasons to be given when the case is reported, the supposition of pretence is about as reasonable as that Brown-Séquard's guinea-pigs should pretend to have fits when the epileptogenous zone is touched.

[2] 'Diseases of Children,' 7th edition.

6. *The Infant's Nervous System.*

The lowest level of evolution is probably nearly the whole of the new-born infant's developed nervous system; the higher levels, in-so-far as they are not yet organised, are masses of "nervous stuff" and not centres proper; they are almost a fœtal part of the new-born infant. No doubt development goes on with increased activity in the higher two levels after birth; the nervous masses, as we called them, will be rapidly becoming middle and highest centres. But their development will be far behind that of the lowest ("earliest") level. "Inward fits" are rare under six months of age, when the lowest level itself will be very imperfectly developed; but probably the main reason why these fits do not often occur until the age of six months is that rickets is rare before that age.

According to Soltmann, the convolutions in the "motor region" (I call this region the middle motor centres) in new-born dogs, cats and rabbits is unexcitable at birth, and epileptiform seizures cannot be provoked in them by excitation of that region. Albertoni has confirmed these observations. Franck and Pitres write of Albertoni's researches: "Il résulte de cette série d'expériences que l'épilepsie par la faradisation de l'écorce du cerveau n'a pu être provoqué chez un chien de treize jours, tandis qu'elle s'est produite chez les animaux âgés de dix-neuf jours et plus." Soltmann's conclusions have, however, been recently disputed by Dr. Joseph Pareth, who says the dog's cortex *is* excitable in the first days after birth. Still we cannot but suppose that the higher centres must, in new-born animals, be a long way behind the lowest in their degree of development.

---

[1] Ringer, 'Therapeutics,' 10th edition, p. 67

[2] 'Lectures on Diseases of Children,' p. 70.

The infancy of a dog in comparison with that of man is of slight duration. The higher the animal, the longer its infancy. Man's infancy is the longest of all. The following quotation from Fiske is of profound importance to the medical evolutionist in many ways.

"The gulf by which the lowest known man is separated from the highest known ape consists in the great increase of his cerebral surface, with the accompanying intelligence and in the very long duration of his infancy. These two things have gone hand-in-hand. The increase of cerebral surface due to the working of natural selection in that direction alone has entailed a vast increase in the amount of cerebral organization that must be left to be completed after birth, and thus has prolonged the period of infancy."[1]

Much is said of the "great excitability" of the nervous system of infants. I should not speak so generally, but would say that the lowest level of evolution is in the new-born infant almost the whole of its then developed nervous system, and thus almost the sole part there is to be excitable. Putting this otherwise, the lowest level is, the younger the infant, the more nearly *its* highest level; it is indeed more nearly its highest and lowest at once, although, later in life, on full development of its middle and highest nervous masses into centres proper, that level will be the real lowest. It itself in the infant will be at once imperfectly developed and actively developing, and thus *naturally very excitable*. The parts most actively developing on the infant's lowest, his then nearly highest, level will be the centres for organic parts; the centres for animal parts will be far behind them in degree of development. No doubt the order of degree of development of the centres for the three great organic functions, although nearly contemporaneous, will be, both before and after birth, digestive, circulatory, and respiratory; the respiratory will be, of the three, the most actively developing in infancy. The lowest centres for the three systems are seated in that part of the lowest level which is, morphologically, the medulla-oblongata, and as Lauder Brunton, in some very important remarks, says: "the medulla oblongata in the vertebrata may be looked on as a lower and more fundamental centre than the brain or spinal cord."[1]

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[1] 'Destiny of Man,' p. 54. For a full account of Fiske's opinions on the importance of prolongation of infancy, see his 'Cosmic Philosophy,' vol. ii.

Another side of some of the foregoing statements is that the higher levels being in the infant little organised (being there morphologically, but not being there functionally to any great extent) the lowest level will be less "controlled" or less "kept down" than in older persons. *Pari passu* with the later development, or evolution, of the higher levels, the lowest will be more and more "kept down." Putting this otherwise; the process of evolution is not only an "adding on," but is thereby, at the same time, a "keeping down;" the newly added (properly the higher and newer *evolved out of* the lower and older) controls its lower. Using popular language, we gradually get above our mechanical selves as evolution of our middle and highest centres (and especially their internal evolution) proceeds.

Here I refer to Soltmann's important researches on imperfect development of inhibitory arrangements in very young dogs, cerebral, he thinks, as well as spinal. If Soltmann's observations are correct, they are a very important contribution to the doctrine of evolution of the nervous system. The younger the infant, the more of a mere ordinary reflex mechanism will be what there then is of its nervous system; the less check there will be of one part by another. The accelerating cardiac centres will develop ahead of the cardio-inhibitory centres; the respiratory ahead of its checking nervous arrangements, the vaso-motor ahead of the vaso-dilator nervous arrangements.

7. *Recapitulation.*

So now we find that "Inward fits" occur in the very young, in those persons that is, whose lowest level of evolution is the most developed and yet most actively developing level, and which, from the comparatively little development of higher centres, is little controlled. This is saying that what we call the infant's lowest centres are its *then* nearly highest centres or highest and lowest at once. "Inward fits" occur in those persons whose *then* nearly highest centres are *naturally very excitable.* Bearing in mind Soltmann's observations, we add that these nearly highest centres (which later in life will be the lowest centres) are more nearly ordinary reflex centres, are less checked in their actions and interactions by inhibitory nervous arrangements. But why have some and not other infants "inward fits"? In other words, what is the determining cause of such discharges of the infant's "naturally very excitable" respiratory centres as produce, not respiratory movements, but respiratory convulsions? That inward fits nearly always occur in the rickety is now (Elsässer, Jenner, Gee) accepted doctrine. But what is the relation? Henoch writes: "All attempts fail to explain the connection between rachitis and spasm of the glottis."[1] It is then somewhat rash in me to submit an explanation. The question is, "By what steps does rachitis bring about sudden and excessive discharges of the respiratory centres in infants? What is the intermediary between rickets and Inward fits?" As a preliminary, some general remarks on nervous discharges must be made.

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[1] 'Pharmacology,' &c., p. 197.

8. *Digression of Nervous Discharge.*

All movements are the results of nervous discharges (or liberations of energy by nerve-cells), but in the case of convulsion there are sudden excessive and rapid discharges; *such* discharges of any motor centre do not produce movements proper.[1] Besides developing movements strongly, they develop many together—produce that contention of many movements which we call convulsion. Any motor centre suddenly, excessively and rapidly discharging is, for the time being, a "convulsive centre." What I call "discharging lesions" in epileptic and epileptiform seizures are small persistent "convulsive centres." A "discharging lesion" (or part of a motor centre become a "convulsive centre") is a local persisting hyperphysiological state of nerve-cells induced by some pathological changes, which changes lead to increased, but yet, I think, inferior nutrition.[1]

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[1] Many years ago Moxon remarked, "muscle is only in degree less nervous than nerve." There is, in severe epileptic fits, a sequence of discharges of four levels of motor evolution—highest, middle, and lowest nervous centres and of "muscle centres"—muscles being in one aspect, in accord with Moxon's remark, nervous centres; in another aspect they are glands. I continue, however, to speak of three levels of evolution.

It is to be insisted on that convulsion, in any case, is nothing whatever else than a sudden excessive and rapid development of the *normal* movements which the centre, suddenly and excessively discharging, represents. The great thing to bear in mind is, that such discharges lead not only to excessive, but also to contemporaneous development of many movements, so that the name "movements" is no longer properly applicable to the motor affection produced, and we call it convulsion. Let any motor centre be discharged strongly enough, and rapidly enough, and we have convulsion of the parts the centre represents.

There is, of course, something more. Since a "discharging lesion" is a physiological fulminate (normal cells of a centre having become a quasi-parasitical hyper-functioning part of it), the sudden excessive and rapid discharge of its *highly* unstable elements upsets the equilibrium of (discharges) anatomically connected nervous arrangements of normal instability; normal elements which, in the case of a "destructive lesion," serve towards compensating a loss, are in the case of "a discharging lesion" compelled to co-operate in an excess (co-operation in excess is compensation inverted). So it is always to be understood, when not stated, that not only are the movements of the parts most especially represented by cells constituting a "discharging lesion" developed, but that the movements of other parts also, represented by normal cells which are (by collateral and downward currents) compelled to discharge, are developed. (The discharges in both cases being sudden, &c., what we just called the development of movements, is really the "running them up" into convulsion.) Hence from sudden and rapid discharges of a small part of the highest centres (in epilepsy proper) we may have from wide collateral and downward discharges universal severe convulsion (co-operation in excess).

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[1] I have been said to have put forward the "theory of discharges" as the *pathology* of epilepsies. I have really tried hard to show that certain pathological processes only *induce* "discharging lesions" which are *hyper-physiological* states.

Convulsion, although very different from a series of normal movements, is not something altogether *sui generis.* I do not now believe, as I once said, that all metaphysic has died out of the expression "attacked by convulsion;" it is not always thoroughly realised that a convulsion is simply a brutish development of many of the patient's ordinary movements. A severe epileptic convulsion, to take that case for illustration, is nothing whatever else than a sudden, excessive, rapid, and consequently a nearly contemporaneous, development of many of the patient's common-place daily movements—of movements of the eyes in looking; of the face in its various expressions; of articulatory movements; of manipulatory and of other movements of the arms; of movements of the trunk and legs in walking; of digestive, respiratory and circulatory movements, &c. But from such developments there is no looking, smiling, talking, &c., because all the movements which are developed, being nearly contemporaneously developed, are "run up" into a muscular fight. Speaking figuratively, we may say that the epileptic discharge is trying to develop all the functions of the body excessively, and all at once; a severe fit is a fairly successful attempt. Let me give a very simple illustration. If there be a centre for locomotion, then during slight sequent discharges of its elements in health, there is walking or running; but if very many of those elements were to discharge suddenly, rapidly and excessively, the man walking or running would not go faster; on the contrary, he would be stopped, would be stiffened up into a tetanus-like attitude by the *contemporaneous* development of many locomotor movements.[1]

We must not speak of "disorderly discharges," but simply of degrees of discharges above the normal—of degrees of suddenness, excessiveness, and of rapidity of discharges. Coming close to our particular topic, we say that whilst normal discharges of the respiratory centres produce a sequence of respiratory movements, sudden excessive and rapid discharges of those centres produce a respiratory convulsion, which is a sudden excessive and contemporaneous development of, and therefore a strong contention of, normal respiratory movements.

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[1] It will be seen that I do not believe that an attack of mania ever replaces an epileptic convulsion; if cells of the nervous arrangements discharging so as to produce movements in mania were to begin to discharge so suddenly, &c., as nerve-cells do in an epileptic fit, the maniacal movements would be "run up" into convulsion.

9. *The Determining cause of sudden, &c., Discharges of Respiratory Centres in Inward Fits.*

It is agreed upon that the normal stimulus to the respiratory centres is venous blood. It has long been recognised that rapidly induced asphyxia in animals produces a certain kind of convulsion, a respiratory one. I believe the determining cause of the sudden excessive and rapid (convulsion-producing) discharge of the respiratory centres in cases of "inward fits" to be an excess of their "natural stimulant," venosity of blood. There is no persistent "discharging lesion," as I believe there to be in epileptic and epileptiform seizures. The normally "very excitable" respiratory centres are occasionally goaded into excessive activity. It is not necessary for me to deny, and I do not deny, that besides being naturally very excitable, the centres developed in the infant, and especially those most actively developing, are *morbidly* over-excitable in rickets and in some other diseases. But I make no use of that hypothesis. But how comes about this excess of the "natural stimulant"—excess of venosity in the rickets?

Mentioning the circular shape of the young child's chest (its respiration being chiefly diaphragmatic), I remark next that "inward fits" occur, not only in persons with such an imperfectly developed respiratory apparatus, but, as is well known, most often in those of them who, from rickets, have abnormally soft ribs. The ribs being abnormally soft in the rickety, and thus not "holding out" during the descent of the diaphragm, the efforts of that muscle are more or less neutralised. Thus there is in young rickety children a condition for venosity (which may in part account for the great sweating in these subjects). Further, the attacks of laryngismus occur chiefly at night when, from sleep, there is still less perfect respiratory action and thus a condition for still greater venosity. A crying fit may bring on a paroxysm (as, anticipating, laughing may bring on an attack of spasmodic asthma in an adult). Henoch[1] writes in his articles "Spasm of the Glottis," "In fact, many characteristics of the affection can be seen in a *healthy* child who, in the midst of a severe crying spell, suddenly 'holds the breath'; *i.e.* lies with the head thrown back, face dark red, somewhat cyanotic, respiration interrupted, and rigid limbs. The excessive crying and the excitement appear to cause spasm of certain respiratory muscles, which disappears, as a rule, after a few seconds." I submit that the thing first caused by the crying in such cases is super-venosity, and that this, over-stimulating the respiratory centres, causes respiratory spasm. Henoch then goes on to speak of *abnormal* conditions: "attacks [of laryngismus] may occur during complete quiet, and *even on awakening from sleep;* but even then it is favoured *by every respiratory exertion,* viz. crying, drinking, eating as well as by psychical influences, anger and fright." The parts I have italicised in the second half of the quotation seem to me in favour of my hypothesis.

Great venosity of the blood will much over-stimulate the "naturally very excitable" respiratory centres in infants, and thus produce respiratory convulsion.[2]

No doubt the respiratory centres are also acted on indirectly through the vagi, the venous blood stimulating the endings of these nerves in the lungs. For when the vagi are cut across, respiration is slower, but yet deeper. Stimulating the central end of one of the vagi, quickens respiratory movements; indeed great stimulation so accelerates the rhythm, that it ceases to be rhythm, ordinarily so-called; there is tetanus of the diaphragm, and standstill of respiration in an extreme inspiratory phase. Here we see that, increasing the rapidity of the discharges of the respiratory centres fuses the respiratory movements into convulsion.

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[1] Op. cit., p. 70.

[2] Possibly, in so far as the higher levels are developed in infants, sleep, which in all persons is, among other things, the going out of function of the highest nervous arrangements (a normal dissolution), leaves the respiratory and other lowest level centres less controlled, and thus more excitable than during waking.

Of course no one can ignore that there is very great venosity of blood in the paroxysm of laryngismus, but my contention is that too great venosity is the first thing, that it precedes any part of the paroxysm. Whilst healthy breathing depends on slight venosity—the respiratory centres acting in healthy states from some slight deficiency of oxygen, or possibly, as some think, from stimulation by carbonic acid,[1] too great venosity will stimulate the respiratory centres so overmuch as to produce, not a sequence of normal respiratory movements, but that excessive development and contention of them which is convulsion—will produce arrest of respiration by fixation of the respiratory apparatus. In healthy respiration the inspiratory part is chief; expiration being to a great extent recoil from inspiration. But even if we could conceive that the inspiratory movements were alone suddenly, &c., developed, the chest would be more or less fixed, fixed in a high inspiratory attitude. In dyspnœa, the expiratory movements come into greater prominence.

When there has been first set up any degree of spasm of any part of the respiratory apparatus, the venosity will become greater still, and thus there will be a rapid multiplication of effects. There are degrees of "inward fits" from that local convulsion signified by "crowing inspiration" (perhaps after temporary complete closure of the glottis), to universal convulsion probably consequent on prolonged closure of the glottis, or on partial closure of it with also fixation of other parts of the respiratory apparatus. The supposition is, that the more rapid the initial discharges (that is, the more elements of the respiratory centres suddenly discharging at once, and thus the greater the rate of transfer of energy), the more is it likely for the convulsion to become universal.

That the convulsion, even when universal, is not owing to discharge of higher levels (not of the so-called motor region, middle level, at least) is proved by the fact, that asphyxia renders the "motor area" of the cortex inexcitable.[1]

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[1] Brunton writes (Pharmacology, &c., Ed. i. p. 202): "It is uncertain whether the stimulation which the venosity of the blood produces is due chiefly to the absence of oxygen or to the presence of carbonic acid. Possibly also it may be due to the products of imperfect combustion in the venous blood. Or all these three causes may share in the stimulation, though to what extent each does so is not known. According to Bernstein, want of oxygen appears to stimulate the inspiratory, and the presence of carbonic acid to stimulate the expiratory centre."

10. *Universalisation of Convulsion beginning Respiratorily.*

Supposing, then, the first sudden, &c., discharge in Inward fits to be of the respiratory centres in seizures becoming universal, the universalisation of the convulsion may be owing to secondarily induced discharge of other centres on the lowest level. Nearly all the muscles of the body must on demand, so to speak, serve in respiration. This implies that the respiratory centres, whilst representing some movements of the nose, larynx and chest nearly directly, represent also movements of nearly all other parts of the body with various degrees of greater indirectness; means that they can "call on" other motor centres to act with them in a certain order; means that their discharges, when strong enough, "find their own" in other centres—flow in lines which are of least resistance *to them*. It is not then necessary to suppose that there are big permanent respiratory centres for all parts of the body, but to hold that there are centres representing most especially the respiratory apparatus, from great over-action of which centres other centres, not ordinarily respiratory, can be called out as reserves, called upon to suspend their own individual duties to serve respiratorily, to join in the fight for breath. The term "centre," in any case, is an arbitrary expression.[2]

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[1] It may be said that some clinical facts show that great venosity of the blood, in adults, "excites the brain." In some cases of emphysema with bronchitis, the patient may be delirious at night. To take a stronger case. A man was recently admitted into the London Hospital under Mr. McCarthy's care, for injury of the spine, which reduced the patient to diaphragmatic respiration. He became delirious, when he was evidently undergoing asphyxia. For my part I cannot see how these facts show that venosity *excites* the brain. I should say that the venosity accounted for the negative half only of the patient's condition when delirious—for the physical condition answering to his defect of consciousness—that it put out of use some of the highest nervous arrangements of the highest centres. I submit that the positive part of the condition, the delirium proper, was the outcome of raised activity of lower nervous arrangements of his highest centres, consequent on loss of control. We remarked before that Evolution is not only an "adding on," but a "keeping down" also; so in the case of Dissolution from venosity just mentioned, the "taking off" of the highest nervous arrangement is a "letting go" of the lower.

[2] For admirable remarks on the term "Centre," see Lauder Brunton, 'Pharmacology,' &c., 1st ed., p. 199.

I refer to remarks made in the section *Digression on Nervous Discharge* on secondary discharges of normal elements consequent on a primary sudden, &c., discharge of highly unstable elements.

It may perhaps be that the same cause, super-venosity, acts directly on other lowest centres, discharging them after discharging more or less of the respiratory centres. Indeed there certainly is action on some other lowest centres; for in artificially produced asphyxia there are discharges of the vaso-motor and sweat-centres, as well as of the respiratory centres.

The presumption is that there are the two effects; that universalisation of the convulsion depends partly on discharges spreading from the primarily discharging respiratory centres to other centres on the lowest level, and partly on elements of these other centres being also discharged by the direct action of over-venous blood upon them.[1]

In the foregoing is a detailed acknowledgment of what was stated generally, in saying that the convulsions in the respiratory fits of children sometimes become universal. There are effects produced by asphyxia in many centres, if not in centres "all along the line" of the lowest level. In children we see turning up of the eyes; asphyxia produces sweating, raised arterial tension; there are carpo-pedal contractions as well as the severer universal convulsion. Hence severe respiratory seizures, there being universal effects, are more thoroughly comparable and contrastable with severe epileptiform and epileptic seizures, in each of which also there are universal effects, than the too limited name "respiratory" I give to them implies.

If venosity in the rickety causes the respiratory fits we call laryngismus in infants, other causes of venosity in very young children should do the same.

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[1] What we have so far called *the* respiratory centre is only the dominating respiratory centre. If the spinal cord be divided just below the medulla oblongata, respiration usually ceases entirely. But if strychnia be injected into the blood before the division, respiratory movements continue to some extent, and begin again if strychnia be injected after the division.—Lauder Brunton, op. cit. p. 201.

11. *Respiratory Convulsions in Laryngitis of Young Children.*

I refer to convulsions in young children, subjects of laryngitis ("spasmodic laryngitis"); they are respiratory convulsions, but may become universal. I suggest that the age of the patients, the fact that there is a manifest impediment to respiration at the gateway of the respiratory apparatus, and the occurrence of the attacks mostly at night (in sleep), point to an over-stimulation of the respiratory centres by too great venosity. If so, the hypothesis that attacks of laryngismus are similarly caused is favoured. "Spasmodic" laryngitis usually occurs in children under two or three years of age; the attacks usually occur suddenly during sleep, and next day the child may be seemingly well except for a little hoarseness and cough; the attacks are sometimes mistaken for laryngismus.

12. *Treatment of Inward Fits.*

Let us now see if the results of treatment favour the hypothesis put forward. It is good practice not to let the child who is the subject of laryngitis sleep too long at a time—not to let it remain too long in a condition favouring venosity; I suppose the same applies to laryngismus stridulus, to which morbid affection I confine further remarks on treatment.

It is understood, of course, that attention is given to diet, digestion and state of the bowels. No one fails to treat the rickets, the most approved medicine being cod-liver oil. The good effect of the highly valued remedy, cold sponging, favours my hypothesis; manifestly it will stimulate the respiratory centres. So do the good results of carrying the child out even in cold weather, and change from town to country air. The several remedies will tend to reduce venosity. Now for drugs, more especially in the treatment of what we may call laryngismus itself. The best remedies should, on the hypothesis, be those which stimulate the respiratory centres. Eustace Smith writes:[1] "Of special drugs, musk and belladonna are most useful [in laryngismus stridulus]. The former can be given to a child of twelve months old in doses of one-third of a grain every six hours, and will be found to have a powerful influence in checking the tendency to spasm. Belladonna, to be of service, must be given in sufficient doses. A baby twelve months old, will take well fifteen drops [of the tincture] three times in the day." Musk[1] is a stimulant to the respiratory centre, as is also belladonna. It is well-known that children tolerate large doses of belladonna; bearing in mind Soltmann's researches, this may be because in them it acts more as a stimulant, or rather that the parts it can render negative (inhibitory and secretory nervous arrangements) are not largely present in the infant to be paralysed.

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[1] 'Diseases of Children,' p. 273.

What is said of the beneficial effect of the stimulating action of belladonna on the respiratory centres is not inconsistent with the hypothesis, that great excess of the "natural stimulus" (venosity) produces the fits; for to keep respiration going on actively will prevent great venosity. But anything which makes the respiratory centres act excessively, causes so rapid a succession of impulses from them to respiratory muscles, as to fix the chest in one persistent inspiratory condition, and brings about next similar impulses to expiratory muscles. Thus there results a fight in which the chest is still in infinite effort, as two well-matched wrestlers are at a certain stage of their contest, the full power of each being developed.

Chloral is a remedy for fits of laryngismus lauded by high authorities. It is the best remedy for a rapid succession of fits of any kind—for putting an end to the seizures, that is. It is not, I presume, actually curative of the morbid condition inducing them. It may be given when fits of laryngismus are frequent.

Now for treatment after the paroxysm. When the fit is over, the child usually comes round spontaneously. But supposing that after a severe attack respiration no longer goes on. The respiratory centres are exhausted, or what remains of them unexhausted is not enough to produce respiratory movements; the child is seemingly dead. There is paralysis of the respiratory apparatus after respiratory convulsions, just as there may be of a limb after an epileptiform convulsion which had affected that limb first and most.[1] Here we must bear in mind that, after asphyxia fits produced in lower animals, the heart, as a rule, continues to beat for a short time after respiration has stopped. Hence if luckily present when a child is seemingly dead after a fit, we should give him a chance of recovery by artificial respiration. We should not waste time to see whether the heart has or has not stopped. The following quotation[2] justifies the above remark, and is of value in several other ways. Brunton is speaking of asphyxia fits artificially produced in lower animals. The first paragraph in the quotation refers to the paroxysm, the second to the after-paralytic stage, the third is directly relevant to what was just said on attempts to restore infants apparently dead after their respiratory convulsion.

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[1] Brunton's 'Pharmacology,' &c., p. 988.

"As the blood becomes venous, the activity of the respiratory centre increases, the respirations becoming quicker and deeper, and the accessory respiratory muscles are thrown into action. This condition is called *dyspnœa*. Finally, the excitement extends to all the muscles of the body, and we get general *convulsions*, which have generally an opisthotonic character. The eyeballs very often protrude during these convulsions, and the blood-pressure rises greatly from stimulation of sympathetic and vaso-motor centres in the medulla.

"After the convulsions cease, the animal usually lies motionless, and the heart, as a rule, continues to beat for a short time after the respirations have ceased.

"The excessive venosity of the blood in this condition has paralysed the nerve centres; but if artificial respiration be now commenced, and the blood becomes gradually aërated, the conditions just described are again passed through in the reverse order; convulsions first reappearing, then dyspnœa, next normal breathing, and if the respiration be pushed far enough, apnœa."

I say nothing further here of carpo-pedal contractions which so often complicate laryngismus; their interpretation is difficult. If we take a mere general view of them, as "spasms," there is no difficulty; but the question is, "Why are the most terminal parts of the limbs so specially subject to spasm in this disease?"

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[1] I believe there to be paralysis after epileptic fits also; but, so far as I know, no one agrees with me in this supposition.

[2] Brunton, op. cit. p. 202.

13. *On Convulsions in some other Morbid Affections of Young Children.*

Besides convulsion in rickety infants, and in those infants whose blood-aëration is more acutely in arrears from catarrhal laryngitis, I suppose that in some other diseases in which there is great venosity in very young children there are respiratory convulsions. Probably convulsions in young children who have congenital heart-disease are of that nature.

Eustace Smith writes,[1] dealing with congenital heart disease; "Convulsions are very common, especially in infants; and startings and twitchings during sleep are seldom absent, whatever be the age of the patients.

"Nearly one-half of the cases die before they have completed the first year, and two-thirds before they are two years old. Death often occurs in a convulsive fit; and infants usually die in or directly after such a seizure."

We have convulsions in some cases of whooping-cough, if indeed the ordinary attacks are not allied to convulsion. Judging from what Henoch says,[2] I should say that some convulsions in patients who have pertussis are very closely like the severe convulsions of laryngismus. The parts I have italicised in the following quotation are particularly interesting in this regard—the first as to age; the second as to contractions, resembling, if not identical with, carpo-pedal and other contractions. Henoch calls attention to "the unfavourable significance of those paroxysms [of whooping-cough], which are characterised by protracted apnœa [?], in which cough, with very few or no inspirations, is alone observed, and therefore no whooping tone is heard. *Such attacks are chiefly observed in little children during the first year of life.* The cyanosis rapidly reaches its highest grade; the suffocation is threatening, and may indeed prove fatal, especially when the disease is complicated with diffuse catarrh or broncho-pneumonia. *Under these circumstances partial spasms (deviations of the eyes, contracture of the fingers, toes, arms, &c.)*, or even *general and fatal convulsions*, may occur during the attack, or immediately afterward, either in consequence of the protracted venous stasis in the brain, or the accumulation of carbonic acid in the blood, which must ensue in the absence of sufficient inspirations."

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[1] Diseases of Children, p. 510.

[2] Op. cit p. 173

14. *On so-called Excentric Irritations; Excess of "Natural Stimulants"; Epileptogenous Zones.*

I have said nothing so far of excentric irritations in cases of laryngismus, not being convinced that they have any share in the production of the seizures. I except, of course, that "excess of the natural stimulus" of the respiratory centres which[1] may consist in strong excitation of the vagal ends in the lungs. Dyspepsia may act, if causing flatulence, by interfering with the play of the infant's diaphragm, and thus indirectly help towards increasing the venosity of its blood. In cases of catarrhal laryngitis there may be excentric irritations from the larynx as part causes of the respiratory convulsions; but if so, here again there is an excess of a natural stimulus, although of a different kind from venosity. I should doubt whether dyspepsia (vide *supra*) would by "stomach irritation" cause any kind of fits. What I find hard to believe is, that excentric irritations of any sort, of any part, can act on "the nervous centres" when they are healthy, so as to produce convulsion. I can understand that convulsion may result if the peripheral part irritated is particularly related to (specially represented in) the centre which is compelled to discharge—is its "natural stimulus"—but not then unless the centre is from some other cause already morbidly over-excitable. The part of the periphery (ento or epi) especially united by afferent nerve-fibres to any motor centre, by the intermediation of sensory centres, is, when that centre has become by disease a "convulsive centre," its epileptogenous zone. I can easily understand, to illustrate by epileptiform seizures, that if a man have already a "discharging lesion" of part of the so-called "arm centre," a tap on some of the tendons of his hand may *start* a fit. And I do not deny that if a man has epileptic fits (epilepsy proper) with a "stomach warning," indigestion may provoke a fit by producing stomach irritation—the stomach being the epileptogenous zone in that patient. On this topic more later. We next consider cases of respiratory fits, experimentally produced in healthy lower animals, when such excentric irritations need not be invoked, even as part causes. This will be the subject of a future paper.

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[1] Of course, excitation of very many parts of the body (nose in sneezing, ear in producing cough, sudden application of cold) act on the respiratory centres. I speak of more definite or particular stimuli, and of excessive stimuli.

ON A CASE OF LOCOMOTOR ATAXIA WITH LARYNGEAL CRISES, AND ONE OF PRIMARY SCLEROSIS OF THE COLUMNS OF GOLL, COMPLICATED WITH OPHTHALMOPLEGIA EXTERNA.

BY JAMES ROSS, M.D., LL.D., F.R.C P.

Case 1.—W. W., aged 35 years, fishmonger by trade, was admitted to the Manchester Royal Infirmary on Oct. 15th, 1884, under the care of Dr. Ross.

History.—The patient has been much exposed to cold and damp in following his occupation, but he has always had an abundance of good food and warm clothing. His family history is good, and he has himself never suffered from any serious disease up to the date of the present illness. At twenty years of age, the patient was unsteady in his habits, and gave way for two or three years to alcoholic excesses, but since that time he has been a temperate man. In 1870 the patient contracted a sore on the penis, and some weeks afterwards the glands of the groin were swollen, and he subsequently suffered from sore throat and a cutaneous rash. During that time he was under continuous treatment for five or six months; but does not know the nature of the drugs which were prescribed for him. Two years after contracting this sore, the patient married, and eighteen months afterwards his wife suffered from a cutaneous eruption, and her hair fell off. His wife has had one miscarriage, and three still-born and four living children; but of the latter two died from convulsions in infancy, and two are still living and moderately healthy.

The present disease began three years ago, by frequently recurring paroxysms of severe pain in the stomach, which were attended by retching and vomiting of large quantities of bile. The attacks recurred almost every morning at first, and sometimes continued during the whole day, but for the last eighteen months they have only averaged about one in a fortnight.

The patient was also "nervous" in the dark, and six months from the commencement he fell forward one morning into the wash-basin, on covering his face with his hands whilst washing. About this time the patient was sitting one day for some time with his legs crossed, and on attempting to rise he felt a severe tingling in the left foot and leg as far as the knee and found himself quite unable to walk, but he recovered the full use of his limbs in a fortnight.

Coincidently with the appearance of the gastralgic attacks the patient suffered from double vision, and he continued to be troubled with this symptom occasionally for some time; but it was not until fifteen months before admission that he observed the presence of a definite squint, which was accompanied by drooping of the left eyelid. From that time up to a few months ago the left eyelid remained almost completely closed; but during the last few months a considerable improvement has taken place in this respect. About the same time that the eyelid began to droop, the patient noticed a decided failure in the acuteness of his vision, which has progressively increased, until he is now nearly blind, especially on the right side. About two years before admission, the patient began to experience a crowing noise along with inspiration. He suffered almost continuously from this noisy inspiration, but in addition he had paroxysms of difficulty of breathing, each of which lasted several minutes. During these attacks he felt as if he were about to choke, and every inspiration was accompanied by a very loud crow, like that of whooping-cough. The patient states that he was told that the noise which accompanied breathing was always present in a marked degree when he was asleep, and during a residence of a few weeks in the Salford Hospital, some months before he was admitted to the Royal Infirmary, the nurse was in the habit of rousing him several times every night, because his noisy breathing prevented the other patients in the ward from getting to sleep. He suffered occasionally from a girdle sensation on a level with the epigastrium, and had some numbness in his feet; but he appears to have been singularly free from lightning-pains, and up to a few months before he came under observation he had no great difficulty in walking, except in the dark. During the last two or three months his power of walking rapidly failed, and now he is quite unable to stand. In the early part of his illness the patient observed that he could go a whole day without having any desire to pass water, but on emptying his bladder as a matter of routine at night, he voided a large quantity. This tolerance of the bladder soon gave place to irritability of it, and for some time he has been compelled both to micturate frequently and to obey the call promptly, but, notwithstanding all his precautions, he occasionally dribbles. He suffers much from constipation, and is not always conscious of it when his stools are passed, while he has occasionally soiled the bed.

Present Condition.—The patient lies on his back, and although he can move his legs about freely when laid down, he is quite unable to stand. He is a thin, spare man, and considerably emaciated, but none of the muscles appear to have undergone special atrophy. The patellar-tendon reactions are absent on both sides, ankle-clonus cannot be elicited, and there is no muscular tension or contractures. The patient states that both feet feel numb, and the prick of a pin does not give rise to much pain in the skin below the knee, while several seconds intervene between the prick and the recognition of the pain. Two points have to be separated about eight inches before they are felt as separate over the skin on the outside of the leg. The patient has also a very imperfect appreciation of the position of his legs when his eyes are closed, and the muscular sense, as tested by different weights attached to the legs, is very deficient. Sensation, as tested by touch and pricking, is impaired in both hands; the loss of sensibility being specially marked in the ulnar half of the hand and two and a half fingers. On being asked to touch with closed eyes the point of his nose by the tip of one of his index-fingers he touches wide of the mark, the finger sometimes hitting one of the eyes instead of the nose. The patient fumbles a good deal when buttoning and unbuttoning his clothes, and he experiences considerable difficulty in seizing small objects. Both eyelids are somewhat drooping, but the left is affected to a much greater extent than the right; and the cornea of each eye is drawn somewhat towards the outer canthus, owing to a predominance in the action of the external rectus muscle, and consequently the patient has a double divergent squint. All the movements of the eyeballs, however, are much restricted, and even outward rotation is not carried out to the normal extent, while the power of rotating the eyeballs upwards and downwards is almost entirely lost. Inward rotation is effected better on the right than on the left side, and indeed the paralysis of all the muscles is less marked on the right than on the left side. The pupils are always moderately dilated; but, as a rule, the right is not quite so large as the left. The pupils fail to contract to the stimulus of light, and they only contract to a slight extent to accommodation. The patient can count figures held up before him with either eye, but he is quite unable to read, having a difficulty in distinguishing single letters. The optic discs are found, on ophthalmoscopic examination, to be in a state of advanced white atrophy. The patient speaks in a peculiar drawling manner—his voice has a slightly nasal quality, and at the end of each sentence the drawing in of the breath is accompanied with a slight noise, like that caused by air drawn through a narrow reed. At night, when the patient is asleep, the noise which accompanies inspiration is said to be much louder than when he is awake; and the nurse states that the other patients near him are frequently disturbed in their rest by it. The patient has to keep a urinal in constant use, and occasionally he has soiled the bed by an unconscious passage of fæces. He was kept five weeks in the Royal Infirmary, during which he was treated with rest in bed, ten-grain doses of iodide of potassium three times a day, and good diet. He was sent to the Convalescent Hospital at Cheadle on Nov. 22nd. Dr. Wansbrough Jones kindly took the following notes for me:—

Jan. 31, 1885.—For the first few weeks after the patient's admission at the Cheadle Convalescent Hospital he sat up a great part of the day, but he was quite unable to stand or walk without assistance. Five weeks after his admission he began to be troubled with severe lightning-pains in his lower extremities, and with attacks of vomiting, which recurred almost daily, and were accompanied by severe lightning and girdle pains. Each attack of vomiting lasted from four to six hours, and the patient was only maintained in tolerable comfort by having subcutaneous injections of morphia. Every deep inspiration is accompanied by a stridulous noise, and this noise is particularly loud when the patient is asleep; but in addition he suffers from severe suffocative paroxysms, each of which lasts several minutes, while they recur on an average twice a week. During the attack each inspiration is accompanied by a loud crowing, like that of whooping-cough or of laryngismus stridulus. A laryngoscopic examination shows that the vocal cords do not attain the normal excursion during respiration, and that the chink of the glottis is much smaller than usual during inspiration. About ten days ago the patient had an attack of herpes over the outer surface of the right ear, which lasted nearly a week. About a fortnight ago three large boils appeared over the sacrum, and now ulcerated surfaces are left, the largest of them being about the size of a crown-piece. The disorders of the movements of the eyeballs and pupils, and the other symptoms, have remained much the same as they were when the patient was at the Infirmary.

Feb. 20.—The ulcers over the sacrum became rapidly cicatrised, but soon afterwards the patient was found to have a remittent temperature, and fluctuation was felt over the sacrum; the skin was punctured, and a considerable quantity of pus was voided. He is still suffering greatly from lightning-pains, and gastric and laryngeal crises, and has to get progressively larger doses of morphia in order to make life tolerable. The abscess over the sacrum continues to discharge; temperature is of remittent type; the patient appears to lose strength daily, and to be undergoing progressive emaciation; and he was consequently sent back to the Royal Infirmary on March 4, 1885.

March 22.—On being readmitted into the Infirmary, the patient lay in bed, and had to be kept under the influence of morphia. His stools are often passed unconsciously in bed, and his urine dribbles, so that a urinal has to be in constant use. The urine is alkaline, and contains numerous bacteria, pus-cells, and crystals of triple phosphate, and the temperature varies from 97·5° in the morning, to 101° in the evening, while the abscess over the sacrum still continues to discharge. The lightning-pains, and gastric and laryngeal crises and other symptoms, continue much the same as they were at Cheadle. The patient is discharged on April 8, 1885.

April 27.—At home the patient was attended by Mr. Pilkington, District Surgeon to the Royal Hospital, Salford. Mr. Pilkington informed me that he got gradually weaker from the time of leaving the Infirmary, and he at last died April 24; three days later Mr. Pilkington was kind enough to make a post-mortem examination, and to send me the crura cerebri, pons, medulla oblongata, and spinal cord. These portions of the nervous system I handed over to Dr. Harris, Pathologist to the Royal Infirmary, and, after hardening, he was good enough to cut sections for me. The spinal cord itself was so brittle after hardening, that it was not found possible to make sections of it, but it could be seen with the naked eye that the whole of the posterior columns, and probably also the direct cerebellar tracts, were diseased throughout their vertical extent. An examination of a transverse section of the medulla oblongata, on a level with the middle third of the olivary body (Fig. A, left half), showed that the sclerosis had extended to the thin layer of white substance which lies over the surface of the cuneate nucleus (Fig. 1, G) and which is the upward extension of the column of Goll, to the layer of white fibres which lies to the outer side of the triangular nucleus (Fig. 1, p r), and which is the upward continuation of

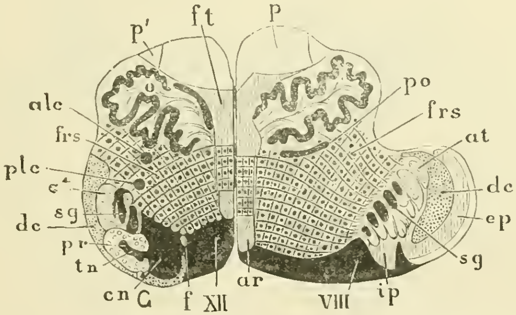

FIG. 1 (modified by Young from Flechsig).—p p,' pyramidal tracts XII-VIII. nuclei of the twelfth and eighth nerves respectively; a r, slender, and p r, cuneate fasciculi respectively; d c, direct cerebellar tracts; a t, ascending roots of the fifth nerves; f, fasciculus rotundus.

the posterior root-zones, and to the layer of white substance which occupies the postero-lateral surface of the medulla oblongata (Fig. 1, d c) and which represents the direct cerebellar tract. Considerable evidences of disease were also manifest in the ascending root of the fifth nerve (Fig. 1, s g), while the fasciculus rotundus (Fig. 1, f) was almost completely disorganised, this tract being traversed by a relatively large vessel with thickened walls, and its substance studded with nuclei, amidst which no healthy nerve fibres could be discovered. A large number of the root-fibres of the vagus were found atrophied. An examination of sections of the crura cerebri revealed considerable changes in the nuclei of the third and fourth nerves and neighbouring parts. The grey substance on the floor of the aqueduct of Sylvius was studded with nuclei, and the ganglion cells of the nuclei were deficient in number, and many of those left were small rounded bodies destitute of processes, these changes being more pronounced in the nuclei of the left than those of the right crus. The fibres of the third nerve, when examined with a high power, could be seen as fine hairs in their passage through the tegmentum and crusta,

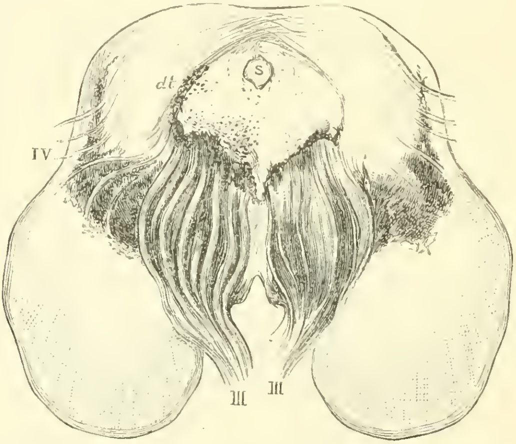

Fig. 2 (after Young).—Section of the crura cerebri. The left lateral half represents the healthy crus, and the right, the crus in the case of W. W.; *d t*, descending roof of the fifth nerve, with fibres from the anterior tubercle of the corpora quadrigemina passing transversely through it; s, aqueduct of Sylvius; III, III, third nerves; IV, fourth nerve.

and the medullary sheath seemed to have disappeared from a large number of the fibres. Scarcely a trace of the bundle of longitudinal fibres which constitutes the descending root of the fifth nerve could be discovered, while the fibres from the anterior tubercle of the corpora quadrigemina which pass through this tract to reach the nucleus of the third nerve were interrupted and atrophied. The annexed diagram (Fig. 2) was kindly drawn for me by my friend Professor Young. The left half of the figure represents a healthy crus, while the right half represents the changes observed in this case. It will be seen that on the right side, as compared with the left, the ganglion cells of the nucleus of the third and fourth nerves are small and few in number, that the third nerve (III) is atrophied, and that the descending root of the fifth nerve (*d t*), with the fibres passing through it from the corpora quadrigemina, has disappeared.

Remarks.—The chief interest in this case centres in the morbid changes found in the cranial end of the central grey tube. In the medulla the principal morbid changes were observed in the white substance of the slender (Fig. 1, G) and cuneate (Fig. 1, p r) fasciculi and the direct cerebellar tracts (Fig. 1, d c), these being respectively the upward continuations of the columns of Goll, the posterior root-zones, and the direct cerebellar tracts of the cord. The fasciculus rotundus (Fig. 1, f) was also found almost completely disorganised on both sides, while minor changes were discovered in the ascending roots (Fig. 1, a t) of the fifth nerves. These bundles are most probably the homologues in the medulla oblongata of the posterior root-zones of the spinal cord, the fasciculus rotundus being associated with the lateral mixed system of nerves consisting of the spinal accessory, vagus, and glosso-pharyngeal nerves, and the ascending root of the fifth being connected, as its name implies, with the great sensory nerve of the head. Changes like those just reported, have already been described by Pierret[1] as occurring in the medulla oblongata in cases of locomotor ataxia, and it is consequently unnecessary for me to make any further remarks upon them in this place. The changes observed in the root-fibres of the vagus in this case corresponds with the cases of tabes with laryngeal crises, reported by Cruveilhier[1] and Jean,[2] in which the roots of the pneumogastric nerves were found atrophied. The most important changes, however, discovered in this case were those found in the crura cerebri. Passing over the morbid alterations of the ganglion cells of the nucleus of the third and fourth nerves, we come at once to speak of those observed in the bundle of white fibres which is named the descending roof of the fifth nerve (Fig. 2, *d t*). This bundle is most probably homologous in its structure and functions with the ascending root of the fifth, which is in its turn, as already stated, homologous with the posterior root-zone of the spinal cord. It is therefore probable that disease of this bundle gives rise to the diplopia and disorders in the movements of the eyeball which so frequently appear in the early stages of tabes, even in the absence of any disease of the ganglion cells of the nuclei of the ocular nerves, although it is highly probable that in the more marked forms of ocular paralysis the ganglion cells are always implicated. It will also be seen from the diagram (Fig. 2) that a small bundle of fibres passes transversely through the descending root of the fifth nerve. These fibres join the anterior tubercles of the corpora quadrigemina with the nuclei of the third and fourth nerves, and it is very probable that some, if not all of them, complete the arcs between afferent fibres in the optic nerves and tracts and efferent fibres in the third nerves, the whole constituting the mechanism which regulates the light-reflex movements of the pupils. The annexed diagram (Fig. 3), borrowed from Erb, shows the nervous mechanism which regulates the movements of the pupil. The light-reflex contraction of the pupil is regulated by the arc (G B C H), passing from the retina (F) to the sphincter of the pupil (E). A lesion in the afferent part of the tract will arrest the reflex contraction of the pupil, but the loss of this reflex will be accompanied by complete blindness; and disease of the efferent fibres (H) will also arrest the reflex, but this loss will be accompanied by dilatation of the pupil and loss of contraction of the pupil to accommodation, because the conducting path from the cortex of the brain at (A) will be injured. But disease of the fibres (Δ) which connect the anterior tubercles of the corpora quadrigemina (B) with the nucleus of the third nerve (C) will arrest the light-reflex, and the loss of the reaction will not be accompanied either by blindness,

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[1] Pierret, 'Essai sur les symptômes céphaliques du tabes dorsalis,' Paris, 1876, p. 19; et, 'Sur les relations du système vaso-moteur du bulbe avec celui de la moelle épinière chez l'homme et sur les altérations de ces deux systèmes dans les cours du tabes sensitif,' Comptes Rendus, 1882, No. 5, 30 Janv. 1882.

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[1] Cruveilhier, 'Anatomie Pathologique,' Paris, 1835–42; Liv. xxxii. p. 19.

[2] Jean, 'Ataxie locomotrice progressive. Troubles ataxiques du côté du larynx et du pharynx,' Gaz. Hebdom. Journ. ii. 1869, p. 121.

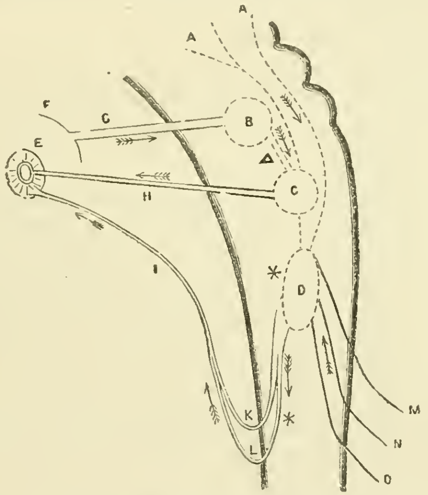

FIG. 3 (after Erb).—A A, psychical impression; B, anterior tubercle of corpora quadrigemina; C, oculo-motor centre; D, dilator centre (spinal); E, iris; F, retina; G, optic nerve; H, oculo-motor (sphincter); I, sympathetic (dilator); K, L, anterior roots; M N O, posterior roots; Δ, seat of lesion causing reflex pupillary immobility; *, probable seat of lesion causing myosis.

paralysis of the sphincter of the iris, or loss of contraction to accommodation. In this case the patient was partially blind, and the sphincters of the pupils were partially paralysed, but neither the blindness nor the paralysis was complete, and consequently the loss of the pupillary light-reflex must have been caused by disease of the fibres connecting the corpora quadrigemina with the nucleus of the third nerve. When,

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[1] For a discussion of the present state of our knowledge of the lighter reflex of the pupil, see Dodds, W. J., "On some Central Affections of Vision;" 'BRAIN,' Part XXXI., Oct. 1885, p. 345.

therefore, we meet with the Argyll-Robertson pupil, it may be inferred that the fibres which connect the anterior tubercle of the corpora quadrigemina and the nucleus of the third nerve are diseased, most probably on both sides; and in locomotor ataxia these fibres are most likely to become secondarily diseased in their passage through the longitudinal fibres which constitute the descending roots of the fifth nerves. It will thus be seen that loss of the light-reflex contraction of the pupil is one of the most surely localising symptoms of disease of the nervous system which we know.

The notes of the following case were taken by my Clinical clerk, Mr. Sidebotham:—

Case II.—D. G., ætat. 35 years, a collier, was admitted to the Manchester Royal Infirmary on April 15th, 1885, under the care of Dr. Ross.

History.—The patient has worked in a coal-pit from a very early age, and has been much exposed to cold and damp, but always lived comfortably. He has indulged pretty freely in alcohol, but he says that he never drank to excess. When a young man he had a sore on the penis, on the glands of which a cicatrix is still to be seen; but he does not seem to have suffered from sore throat or cutaneous eruptions. He has been married twelve years, and his wife had a miscarriage soon after their marriage, but she has had no living children. The patient enjoyed remarkably good health up to the date of his present illness. About three years ago the patient felt for the first time a weakness about the knees, and this gradually increased so much, that at the end of a few months he was compelled to leave off work, and he has never been able to resume it. Until lately he has not suffered from any of the paræsthesiæ, and other sensory disorders which are so common in the first stage of locomotor ataxia, and it is only three weeks ago that he felt for the first time a slight girdle sensation passing round the lower part of the chest. He has not apparently suffered from any lightning-pains.

Present Condition.—The patient is a strong, muscular man, having a dull and sleepy expression, chiefly owing to well-marked double ptosis. The eyeballs are almost completely immovable in every direction; but the pupils, which are somewhat dilated, contract readily both to light and to accommodation. The patient has considerable difficulty in recognising colours, and his vision is somewhat impaired, while an ophthalmoscopic examination reveals a considerable degree of pallor of both optic discs. The patient speaks in an indistinct and drawling manner, and he has to balance his words like a man in the first stage of intoxication. He states that his articulation was at one time very distinct and clear, and that the present defect is of recent date. There is some degree of anæsthesia of the skin of the lower extremities, as high up in the front as the junction of the upper and middle thirds of the legs, and behind as the popliteal spaces. The patient knows the position of his legs accurately when his eyes are closed. The patellar tendon-reactions are completely absent, and no ankle-clonus can be elicited. When his feet are placed side by side along their inner edges, the patient sways a little from side to side on closing his eyes, but with a little effort he can maintain himself erect. In walking, his gait is somewhat straddling, uncertain, and staggering, but not like that of locomotor ataxia. The patient will not walk much without the support of a stick, as otherwise he would, he says, stumble or fall. In walking, his feet are kept widely apart, and the advancing foot is raised to an abnormal degree from the ground; but it is not brought down with a thump, and the heel does not strike the ground first, as in ataxia. The body also rocks a little from side to side in walking, but it is more like the reel of cerebellar disease than the balancing of the body in tabes. The patient says that his knees are liable to give way suddenly, as if his legs were paralysed; and if he were not to exercise great care in supporting himself with the stick, he would often fall to the ground. He was sent to the Cheadle Convalescent Hospital on May 18th, and for the following particulars of his case I am indebted to Dr. Wansbrough Jones.

The notes taken by Dr. Jones confirm on the whole those taken by Mr. Sidebotham at the Infirmary. The only new symptom observed was that during the last few weeks of his life he began to complain of lightning-pains in the lower extremities, but they never attained to any great severity. Notwithstanding his sad condition, the patient was very lively and fond of joking, and had the reputation amongst the patients of being a "romancer;" but there is nothing to show that he ever manifested any of the "grand ideas" which might be considered to indicate general paralysis. On July 13th the patient had just taken his medicine—ten grains of iodide of potassium—and immediately went to the smoke-room, where he began to joke with some of the other patients. In a few minutes afterwards he suddenly became insensible, and was carried to bed. After a few minutes he recovered so far as to ask for a drink, but was soon seized with a second apoplectiform attack, from which he again made a partial recovery, to be almost immediately seized with a third attack, in which he died, the time from the commencement of these attacks till death not being more than ten minutes.

The autopsy was conducted by Mr. Corbett at noon, on July 15th, in the presence of Dr. Wansbrough Jones and myself. The brain was found somewhat congested, and about an ounce of fluid escaped during removal; but no other evidence of disease was discovered, either on the surface of the organ, or after the most careful slicing. The spinal cord, medulla oblongata, pons, and crura cerebri, were reserved for microscopical examination. The heart, large blood-vessels, lungs, kidneys, and all the other internal organs, were found healthy. On examining sections of the spinal cord, some stained by carmine and others by Weigert's method, kindly prepared for me by Dr. Harris, it was found that the columns of Goll were in a state of sclerosis (Fig. 4), throughout their whole longitudinal extent in the cord, but no decided morbid changes could be discovered in the upward continuation of these tracts in the medulla oblongata (Fig. 1, G). But although the disease was most marked in these columns, it was not strictly limited to them in the inferior portion of the cord. On a level with the fourth pair of sacral nerves (Fig. 4. S_4), a level at which, according to Flechsig, the columns of Goll are not represented, the diseased patch occupies a small triangular area, the apex of which hardly reaches as far forward as the middle of the posterior median fissure. The base of the triangle is directed to the posterior surface of the cord; but on this surface the disease extends laterally, and passes along the outer surface of the internal bands of the posterior columns—Burdach's columns—as far as the posterior roots. On a level with a fifth pair of lumbar nerves (Fig. 4, L_5) the apex of the medium triangular diseased area reaches further forwards than in the last section, and on attaining to the level of the third pair (Fig. 4. L_3) it reaches quite up to the posterior commissure; while on the posterior surface of the cord at both levels the disease extends laterally up to the posterior roots. On a level with the first pair of lumbar nerves the disease has the same kind of distribution as on the level of the third pair, except that at the former of the two levels it extends laterally to some slight extent along the posterior commissure, while a small patch of healthy fibres are

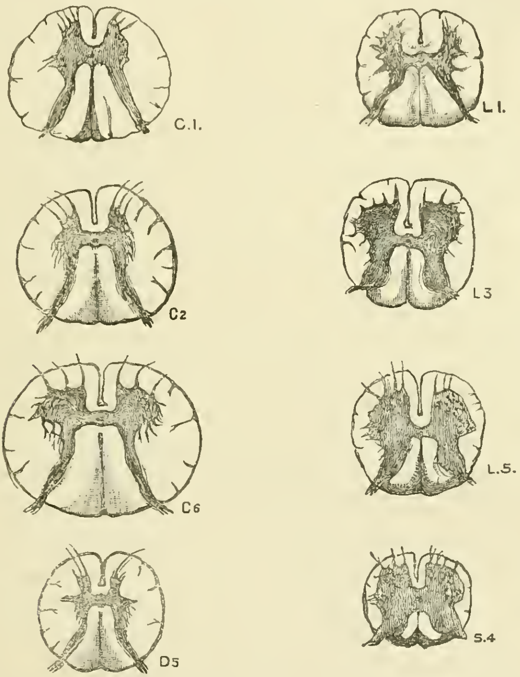

Fig. 4 (after Young).—Sections of the spinal cord from the case of D. G., on a level with the 4th sacral, 5th, 3rd, and 1st lumbar, 5th dorsal, and 6th, 2nd, and 1st cervical pairs of nerves respectively. The shaded portions in the posterior columns represent the transverse distribution of the sclerosis.

observed on each side of the middle third of the posterior median fissure. On a level with the first, second, and third lumbar nerves the posterior roots and posterior horns appear to be implicated in some slight degree in the disease. An examination of sections at different levels in the dorsal region shows that on ascending from the first lumbar pair of nerves the diseased area gradually recedes, so that on a level with the fifth pair of dorsal nerves (Fig 4, D_5) the apex of the median triangle does not quite reach up to the posterior commissure, and the lateral extensions on the posterior surface fall short of the posterior roots, although still invading to a slight extent the external bands of the posterior columns. In the upper dorsal and inferior cervical regions (Fig. 4, C_6) of the cord the disease becomes strictly limited up to the columns of Goll, while in the upper cervical region (Fig. 4, C_2 and C_1) only a small portion of these columns is implicated; but the annexed figures render unnecessary a further description of the distribution of the disease at these levels.

In the medulla oblongata the fasciculus rotundus was probably the subject of a slight degree of sclerosis on both sides, but the ascending roots of the fifth, and the direct cerebellar tracts, were found healthy. In the crura cerebri, the grey matter on the floor of the aqueduct of Sylvius stained deeply with carmine, and when examined microscopically, the tissue at this point was thickly studded with nuclei, while many of the ganglion cells were found to be small rounded masses, destitute of processes. Many of the fibres of the third nerves were extremely fine, and apparently destitute of medullary sheaths. The descending root of the fifth nerve, and the fibres which pass transversely through it, were very distinct, and free from any trace of disease on one side; but on the other side some of the longitudinal fibres had apparently undergone sclerosis, as evinced by a higher staining with carmine, and the absence of the cut ends of the fibres. The diseased fibres were, however, comparatively few in number, and the transverse fibres coming from the corpora quadrigemina were not apparently affected.

Remarks.—The fact, that the disease had extended to the external bands up the posterior columns, renders it doubtful whether this case ought to be described as a primary sclerosis up the columns of Goll; but as it is probable that the sclerosis had not been quite circumscribed in Goll's tracts in any of the other reported cases of this affection, we see no valid reason for refusing to place the case just described under this category. Passing over the cases recorded by Dr. Castel[1] and Gowers,[2] as not presenting any definite symptoms during life which could be referred to the disease found in Goll's columns, we come to consider the classical case reported by Pierret.[3] The drawings which illustrate the report of this case show that the disease of the median columns must have encroached to some extent on the external bands on a level with the twelfth dorsal nerves, much in the same way as it did in the middle dorsal region in my own case. No special mention is made by Pierret of the distribution of the sclerosis on a level with the first and second lumbar pairs of nerves, but a little above the middle of the lumbar enlargement a patch of disease was noticed on each side of, but somewhat removed from, the posterior median fissure, and, if we take Flechsig's diagram as our guide, these patches must have been situated, not in Goll's, but in Burdach's columns. In a case of primary sclerosis of the columns of Goll reported by Kahler and Pick,[4] of which we regret to say we have only seen a short abstract, it is stated that the disease was "tolerably accurately" limited to Goll's columns, and it may therefore be inferred that the morbid changes had encroached to some extent on Burdach's columns. In a case of this affection reported by Camuset,[5] the description shows that the disease had much the same kind of distribution as was observed in my own case, and it is expressly stated that in the lumbar region the morbid changes had extended so as nearly to reach the posterior roots.

[1] Du Castel, "Sclérose primitive des cordons de Goll;" 'Gaz. Méd. de Paris,' 1874, No. 3.

[2] Gowers, 'Lancet,' vol. ii. 1879, p. 876

[3] Pierret, "Notes sur un cas de sclérose primitive du faisceau médian des cordons postérieurs;" 'Arch. de Physiologie,' 1873, p. 74.

[4] Kahler und Pick, "Zur Lehre von der Fasersystemen in den Hintersträngen des Rückenmarks;" 'Prager Zeitschrift für Heilkunde,' Bd. ii. 1881, p. 317. Abstr. Virchow und Hirsch 'Jahresb.,' Bd. ii. 1881, p. 93.

[5] Camuset (L.) "Sclérose primitive des cordons de Goll;" 'Le Progrès Médical,' tome xii. 1884, p. 1008.

A study of the cases collected and analysed by Strümpell[1] and Adamkiewicz[2] shows that the portion of Burdach's columns which are usually spared to the last in cases of ordinary locomotor ataxia—viz. the posterior third and the anterior part adjoining the posterior commissure—were the parts which alone were attacked in the case just reported, thus showing that this case differs greatly in its morbid anatomy from ordinary cases of posterior sclerosis. The presence of ophthalmoplegia externa and the absence of the patellar-tendon reaction in my case, however, show that there is a close relationship between primary sclerosis of the columns of Goll and genuine locomotor ataxia; and it is very probable that had my patient lived longer, the disease would have implicated the external bands of the posterior columns to a much greater extent than it had done, and the case would then have been regarded as an example of the ordinary form of *tabes dorsalis.*

As regards symptoms, Pierret's case suffered from numbness and lightning-pains in the limbs, and girdle-sensations passing round the body; and in Kahler and Pick's case there were first violent lumbar pains and tearing and drawing pains in the legs; but in Camuset's case there was a marked absence of lightning-pains or other sensory disturbance; while in my case there was some anæsthesia of the legs, but the lightning-pains appeared for the first time only a few weeks before the patient died.

The gait of the patients whose cases are reported by Pierret and Camuset was not unlike that of my patient; in all the cases walking was impossible without the aid of some support—a crutch or a stick, and the walk consisted of an uncertain stagger, rather than a genuine ataxia. Loss of the patellar-tendon reactions was not known as a symptom at the date of Pierret's observations, and the state of these reactions is not mentioned in the abstract of Kahler and Pick's paper, or by Camuset; but the reactions were absent in my case. It is probable that loss of these reactions was caused by the slight lateral extensions of the disease to the posterior root-zones at the posterior surface of the cord, rather than to disease of the columns of Goll themselves. The presence of swaying movements on the eyes being closed in this case, tends to show that this symptom is caused in locomotor ataxia by implication of the columns of Goll. This conclusion is, however, rendered doubtful by the fact, that the difficulty of maintaining the erect posture was not increased on closing the eyes in Camuset's case. The activity of the light-reflex of the pupil during life in this case, and the absence of any evidence of disease of the transverse fibres which pass through the descending roots of the fifth nerves, furnish, to some extent at least, the negative side of the proof of the theory, that loss of the light-reflex is caused by disease of these fibres. The symptoms which immediately preceded death in this case, show, like many other reported cases, that a patient may die suddenly with all the phenomena which attend a massive hæmorrhage in the brain, without anything being found after death, either in the brain or other organs, to account for the fatal issue. It is worthy of note that in the case reported by Camuset the disease proved fatal by an acute cerebral seizure. The patient, who had always suffered from a slight feebleness of the mental faculties, was at last seized with a severe attack of acute mania, which proved fatal at the end of twelve days. In the case reported by Kahler and Pick, the patient died from incontinence of urine, cystitis, bed-sores, and septic fever, this being the most usual mode of termination of all chronic affections of the inferior part of the spinal cord. It is difficult to account for the presence of these symptoms unless the disease had extended beyond the area of Goll's columns in the upper lumbar region on a level with the automatic centres of the bladder and rectum. In my case the whole area of Goll's columns was diseased in the dorsal and lumbar regions of the cord, yet the patient did not suffer from any disorder of the functions of the bladder, or from bed-sores.

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[1] Strümpell (A.), 'Archiv für Psychiatrie,' Bd. x. 1880, p. 676; Bd. xi. 1881, p. 27; and Bd. xii. 1882, p. 723.

[2] Adamkiewicz, "Die anatomischen Processe der Tabes dorsualis;" 'Aus dem XC Bande der Sitzb. der K. Akad. der Wissensch.' III. Abth.,' October Heft. Jahrg. 1884.

ON THE RELATION BETWEEN THE POSTERIOR COLUMNS OF THE SPINAL CORD AND THE EXCITO-MOTOR AREA OF THE CORTEX, WITH ESPECIAL REFERENCE TO PROF. SCHIFF'S VIEWS ON THE SUBJECT.

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THE problem which most attracts the attention of all classes of neurologists, whether clinical, experimental, or anatomical, is the identification of function with certain spots in the brain, and discovery of the channels of communication which unite the central nervous system with a harmonious whole. It is the discussion of the relation between the afferent paths in the spinal cord and the motor cortex which concerns us in this paper, renewed attention having been lately drawn to the subject by the recently published experiments of Prof. Schiff. Briefly we may state the case, with a slight preamble, as follows. Every one admits the necessity of regarding the whole central nervous system as being built up on the plan of (1) an afferent channel leading to (2) a sensory centre, which is in close communication with (3) a motor centre from which issues (4) an efferent channel.

Now the bare existence of these constructive factors being granted, it is only their anatomical arrangement which is hotly disputed. There may be said to be three chief views on this subject.

A. The "Separatist" View.—This view places the sensory (perceptive) centres in a distinct, though neighbouring, part of the cortex from the motor centres which are situated in the excitable area of the same. The posterior columns or afferent channels, therefore, are in direct communication with the former of these centres, and only reach the latter (the motor) indirectly by the commissural connections between the centres.

B. The "Fusion" View.—A favourite mode of regarding the subject on the Continent appears to be one in which the sensory (at least that for tactile sensibility) centre is fused with the motor centre. This view, it is to be supposed, carries with it the assumption, that the posterior columns of the spinal cord are in direct communication with the motor cortex.

C. Prof. Schiff's View.—Prof. Schiff states that he has traced (physiologically) the afferent fibres (of the posterior columns of the cord) directly to the excitable portion of the cortex, which he therefore regards as being the sensory perceptive centre for tactile sensibility. From this sensory perceptive area he believes that fibres go down to the motor centres, which are situated he knows not where.

To recapitulate; it is clear that, according to the first view, (2) and (3) are situated in different parts of the cortex, (3) being identical with the excitable area. In the second view, (2) and (3) are fused together in the excitable area; while finally, according to Schiff, (2) is identical with the excitable area, and (3) is somewhere else.

This much being clear, it is now my intention to detail at length Prof. Schiff's mode of reasoning and experimentation, which has led him to the above conclusion. I shall then point out the fallacies in the same, and finally describe fully the experiments I made last autumn to investigate this point. We owe the unusual opportunity of learning much of Prof. Schiff's work to the scientific zeal and energy of Dr. W. R. Huggard, who communicated to the 'Lancet' of Aug. 1, 1885, a paper describing the above-mentioned work, and entitled, "Professor Schiff's Experiments on the Excitable Area of the Cerebral Cortex." In that paper he states pretty fully the way in which Schiff looks at the subject, and his method of investigating the same. Before entering on the practical side of the matter in dispute, I shall criticise the line of reasoning and the points of explanatory meaning, which occur in Dr. Huggard's report.

Such examinatory criticism must necessarily be somewhat disjointed, as the tale which is told in the above-mentioned paper is, in the nature of things, a general essay on the work of the distinguished Genevese Professor. Possibly, however, very little difficulty will be met with in picking up the general thread of the argument. We may note at the outset an enunciation (p. 8 of separately published copy of Dr. Huggard's paper[1]) of the function of the excitable area of the cortex which is intended to be descriptive of the action of a sensory perceptive centre, but which, as a matter of general understanding, is, more properly speaking, a perfect explanation of the function of a motor centre. The passage runs thus: "The grey matter of the excitable area transmutes sensibility into a liberating (probably voluntary) current." The usual interpretation which is put on such a term as "sensibility" is the appreciation by a sensory perceptive centre, of message conveyed thereto by an afferent channel, for consciousness is connoted. Hence Prof. Schiff's real position is that the excitable area is a portion of brain which transmutes the energy received from a sensory perceptive centre into a motor impulse ("*voluntary current*") and that is one which all upholders of the first or separatist view (A. before described) will cordially agree to.

It seems to me very necessary to dwell upon these apparently trivial differences in modes of expression, for it is just ambiguity in the meaning of terms which of itself creates diversity of opinion where very often agreement really exists. To pursue this point a little further, we find that Prof. Schiff (and following him Dr. Huggard too) is disposed to consider that certain apparently paretic (I say apparently, merely for the sake of argument, as I shall show directly the condition is one of real paralysis) conditions are capable of being referred to as ataxia. Perhaps it will be better to illustrate Prof. Schiff's position by the statement of the experimental result on which he bases this opinion. He has found that removal of the sigmoid gyrus, on the one hand, and the effects (after four or five days have elapsed) of cutting the posterior column of the cord on the other, are both productive of similar symptoms, viz. paresis (voluntary paralysis, really), and that this apparent paresis disappears when the animal runs about on a rough surface. The condition following these operations he calls ataxia.

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[1] Wherever paging of Dr. Huggard's paper is mentioned hereafter, it is to be understood as belonging to the Separate publication.

The adoption of this term to express the condition which results from destruction of the sigmoid gyrus might perhaps be justified, if the exactly similar symptom was evoked by section of the posterior columns of the cord only. Prof. Schiff, of course, considers this latter point is established by his experiments; but I shall show directly that (to use Dr. Huggard's words) "they do not warrant the inference," and in fact that "an essential factor has been overlooked." To proceed with the examination of this so-called ataxic condition, it will be well worth while to describe in greater detail the effects of removal of the sigmoid gyrus in the dog, and the more especially since the hiatuses in Prof. Schiff's description, or perhaps I should more correctly say Dr. Huggard's report, happen to include the omission of some of the most characteristic features. Thus the position of the paresed limbs is very constant and striking. The animal stands resting mainly on the limbs of the opposite side, and with the toes of the paralysed side doubled up, so that the dog walks really on the dorsum of the manus and pes respectively. Precisely the same condition sometimes follows section of the posterior columns of the cord, for the simple reason (see p. 5) that the pyramidal tracts frequently degenerate at the seat of operation, and further, exactly the same condition follows section of the principal nerve-trunks in the limb itself. Next, if thorough examination be made of such an animal, it will be found that there is complete paralysis of voluntary movement, the animal is unable to purposely extend the toes and wrist, &c. It should really be scarcely necessary at the present time to point out again the enormous difference in the proportion of cortical representation of the peripheral muscular segments in the dog as compared with other animals, but from internal evidence in the above-mentioned report it does not appear that this fact is fully appreciated yet. The amount of differentiation exhibited by the various so-called centres is extremely small in the dog. Even the extensor muscles of the paw and digits, the representation of which may be considered as the acme of cortical evolution in the dog, can very rarely be called into solitary action by stimulating the cortex even with minimal currents. And just as local differentiation in the extreme point of cortical evolution only crudely exists, so also the extent of the excitable area for the limb movements is extremely small compared with the rest of the brain, hence it is clear that in such an animal the very primitive phenomena of equilibration and progression are most unlikely to be disturbed by removal of the mechanism of those volitionary impulses which occasionally mollify them. To sum up this point, it is clear that whatever part of the volitional motor tract be injured, whether it be the nerves in the limb (provided, of course, all the motor nerves in the limb are not cut, but only those supplying the two main distal segments), the pyramidal tracts in the cord, the internal capsule or the cortex itself, the result is the same as might have been expected. It only remains now to take note of the two points on which most stress is laid by Prof. Schiff, namely, first his assertion, that removal of the excitable area in the dog is followed by loss of tactile sensibility on the opposite side; and secondly, his "crowning evidence" (pp. 6, 7), which consists of the following observation: "In dogs whose posterior columns were cut in the cervical region, if, after the lapse of about four days, the so-called motor region is laid bare, it is found on both sides to be no longer excitable. If one posterior column only is cut, the opposite side of the brain only is inexcitable. If the posterior columns or posterior roots (?) are cut in the lumbar region, irritation of the cortex still produces movements of the anterior limbs, but not of the posterior."

Now as regards the first of these two points, viz. the asserted disappearance of tactile sensibility after removal of the opposite sigmoid gyrus, I cannot speak from personal observation on dogs, and therefore simply refer the reader to Professor Ferrier's 'Functions of the Brain,' pp. 216–217, where this question and that of ataxia too, is sufficiently discussed. We may therefore put aside the first point and take up the second, viz. his attempted demonstration of the direct extension of the posterior columns of the cord to the sigmoid gyrus of the opposite side. His method of experimentation, so far as can be made out from the necessarily short description given in the before-mentioned paper, consists simply of exposing and dividing one posterior column, say the left; then preserving the animal for a period of not less than five days; then exposing the opposite sigmoid gyrus and stimulating it with the ordinary induced current, and observing the absence of the usual movements of the left limbs. The section of the posterior column was usually made opposite, about the fourth cervical nerve, and the motor centres for the fore-limbs of the same side tested as described.

The whole gist of this experiment, as ingeniously designed by Prof. Schiff, lies in the provision of the interval of five days between the section of the cord and the testing of the opposite motor gyrus. For he considered that the consequential ascending degeneration of the posterior column would, in that space of time, reach the opposite cortex and cause degeneration of the grey matter there, which grey matter, we have already seen, he believes to be the perceptive centre of tactile sensibility.

My criticism of this experiment is that it does not justify the conclusions drawn from it by Prof. Schiff until microscopic examination has shown that (1) there is such degeneration of the sigmoid gyrus, and (2) that there are no further changes in the spinal cord than the lesion and degeneration of the posterior column. Feeling the extreme importance of deciding the question thus raised by Prof. Schiff, I determined to repeat his experiment, directing my special attention to these two points; and it will be seen from the results I publish below, that in the first place no such degeneration of the sigmoid gyrus can be demonstrated; and in the second place that Prof. Schiff's "crowning evidence" in support of his position is entirely nullified by the fact, that the section of the posterior column of the cord sets up local degenerative changes in the motor tracts of the cord.[1]

I shall now proceed to expand this latter statement, by detailing the way in which I have repeated Prof. Schiff's experiment and the results I have obtained.

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[1] English neurologists at any rate will be interested to learn (vide 'Lancet' of the 20th of February, 1886) that Prof. Schiff is about to combat the general opinion that the crossed lateral tract is motor.

The animal, a dog, being very deeply[1] narcotised with ether, an incision was made directly over the fourth cervical spine, which being removed, a trephine hole was made through the laminæ of the fourth cervical vertebra. (As a rule the fourth was chosen, but in some of the experiments the spinal canal was opened through the fifth.) The dura mater was then incised by a little transverse cut, and the posterior surface of the spinal cord exposed. After the flow of cerebro-spinal fluid had stopped, the left posterior column of the cord was carefully divided by a small puncturing incision made with a sharp-pointed tenotomy knife. The wound was then closed completely, full antiseptic precautions being taken. On this latter point it is necessary to digress a little. I have performed seven experiments; in two of these the antiseptics failed, owing to the fact, that the animals were so deeply narcotised as to cause cessation of respiration, &c. This necessitated artificial respiration, &c., the spray being discontinued and the windows opened, hence the contamination of the wound. In the remaining five the hole in the spine and dura mater was found by me to be completely plugged, with a dense fibrous plaque of cicatricial tissue, and the soft parts completely united; in two instances the skin being bulged by a collection beneath it of wound serum. In the two instances in which the antiseptics failed, the animals of course suffered from septic meningitis; the subdural space in one being distended with pus, while in the other the arachnoid as far down as the middle of the dorsal region was raised from the cord by pent-up sero-pus. The pus of the wound in each of these two cases was fetid, having a sickly odour, and containing numerous putrefactive bacilli. The advent of this septic condition had only this influence on the experiment, viz. that it made Prof. Schiff's result come out very markedly, the reason of which is obvious.

To return to the method of experimentation.[2] With the two exceptions given above, the animals did not appear inconvenienced, except by the motor paresis, &c. During the five days' interval between the completion of the above-described operation and the exploration with induced current of the two cortices, careful examination was made to find whether there was any motor or sensory paresis to be discovered in the left fore-limb. The testing of the former was of course designed so as to make it clear whether the disturbance of voluntary movement was such as to cause incapability of performing *purposive* acts. As regards sensation, the testing of tactile sensibility was performed in three different ways. In the first place the skin was touched lightly and painlessly by means of a pin; in the second place, I used the spring clip referred to by Prof. Schiff; lastly, I devised as a specially sensitive test the injection of water on to the part (the nerve reaction of which may have been hampered), the sensibility of which was in question. At the outset, I think it very desirable that it should clearly be understood by those who have to estimate the value of original research in this direction, how very difficult it is to correctly gauge the appreciation by one of the lower animals, and especially a dog, of tactile impressions. It is not scientific to draw hard-and-fast lines of distinction between the sensory appreciation of touch, pain, temperature, &c., although of course there is actual difference between the perception of these various physical conditions; in short, since we are usually dealing with combinations of any two or more stimuli —stimulation, as a rule, is compounded of several factors—it would seem only natural that the paths conveying such stimuli are not likely to be very highly differentiated. This non-differentiation is of course simple enough to understand as an outcome of such confusion of sensory function, but it must not be left out of mind when we come to consider the appreciation of such different sensory stimuli as those I have already described. I will now enumerate the facts which I observed in my own experiments bearing on this and the foregoing point.

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[1] On account of the exquisite sensibility of the dura mater.

[2] I desire here to express my indebtedness to my friend Dr. Beevor for kindly assisting me in the record of several of the following observations.

As I have found the affection of sensation to be coupled with the affection of movement (*i.e.* voluntary paralysis, Schiff's ataxia) in a very suggestive way they may, with advantage, be considered together; and as the motor paresis is by far the easier to recognise, and to explain in accordance with the well-understood function of certain tracts in the spinal cord, I shall take that first.

Paresis.—Of the whole number seven of experiments, the question as to whether the condition which I have shown to be perfectly typical of paralysis of voluntary command of the distal segments of the fore-limb existed or not, could only be determined in six, as the first was complicated too much by septic meningitis. Of the remaining six, this paresis was present in three and absent in the rest, although in every case the complete division of the posterior column had been perfectly effected.

Here, then, is a very definite result, which of itself would be quite sufficient to negative the view that the paresis was a form of ataxia; for though the channels of afferent impressions (according to Schiff) had been destroyed in three cases, there was no "ataxy" produced at all. It is clear therefore that the paresis must be due to damage done to a motor tract lying in proximity to the area of section. Now the two well-known divisions of the pyramidal tract, the direct and crossed portions, are both in proximity to the area divided; for while the former, situate in the anterior column, is only separated by the commissures from the posterior column, the crossed lateral tract is still nearer, being only divided from the latter by the slender posterior cornu of grey matter. In every case one or both of these divisions of the motor tract was degenerated (as will be explained directly), in very varying degree. The cases in which no paresis was observable were those in which the degeneration was very slightly marked; while in those instances where it was severe, the paresis was very definite. This fact of "cortical" *i.e.* voluntary paralysis following a localised patch of acute (*vide* infra) degeneration in the pyramidal tract, will not of course be surprising to any one (save those who question the motor function of the pyramidal tract, *vide* note, p. 47); but connected with this observation of the formation of a local block in the motor tract, as a consequence of the division of the posterior column, is the question of the degree and causation of the tactile anæsthesia referred to by Prof. Schiff.

The exact relations, *i.e.* corpuscle and ganglion-cell connections of the fibres of the posterior columns of the cord, are not yet accurately known, all, in fact, that we do know of their function being derived from experiments (artificial and natural) on the effects which follow their damage. Certain it is from such data that if one posterior column be divided, ascending degeneration can be traced from the point injured as high as the bulb. Whether all the fibres in the column are thus disorganised as far as the top of the spinal cord, is another matter, and one which I cannot enter on now. Granting, then, that the fibres of the posterior column are afferent as to the nature of their function, I consider that the results of two of the experiments (viz. III. and IV.) in which there was no appearance of disturbance of tactile sensibility, although the posterior column was most completely divided, and the ascending degeneration very well marked, throw grave doubt on the idea that the posterior columns convey afferent tactile impressions. In further confirmation of this idea, which, resting as it does on two experiments only, is certainly open to the charge of being too slightly founded, I wish to note fully the instances, also two in number (Experiments II. and VI.), where this loss of tactile sensibility was well marked. In these cases (II. and VI.) there was undoubtedly tactile anæsthesia; but there was in addition very definite motor paresis, and of course, as explained above, distinct degeneration in the *lateral* column. Now this couple of cases, II. and VI., presents the exact converse of the condition seen in III. and IV., where the affection of the motor tract was very slight indeed. Hence we are brought to this juncture: Is the loss of tactile sensibility due to degeneration attacking the direct cerebellar tract in the most posterior portion of the lateral column, or to a similar damage of other afferent fibres in the lateral column? It is, of course, quite superfluous to discuss the existence and function of the direct cerebellar tract, but it is worth while to pause for a moment, and to remember that the exceedingly careful experiments of Miescher demonstrated (by accurate measurement of the variations of blood-pressure) that afferent fibres of so-called vaso-motor influence travelled up to the encephalon in the lateral column of the spinal cord. Degeneration, we have just seen, was present in the lateral columns in my experiment when tactile anæsthesia was noted, and again this latter symptom was absent in the cases where the degeneration was scarcely visible. So here we have the crucial test. I cannot help feeling, therefore, that the question as to the path in the spinal cord of the impressions which arouse tactile sensibility must remain undecided.[1]

To sum up. After the above described operation on the spinal cord, *paresis* and *tactile anæsthesia* were only present in those instances where degeneration had attacked the antero-lateral columns.

I will now take up the last division of my subject, and present the results of stimulation of the cortex of the side opposite to the lesion of the posterior column in the spinal cord.

As however the whole seeming abnormal deficiency of function in the excitable area is most obviously due to the degeneration I have referred to, situated in the pyramidal tract opposite the seat of operation and not, as Prof. Schiff would have us believe, in the excitable area, I think it will be best to describe the state of the spinal cord in each of my experiments.

The spinal cord operated on as above was examined in the the following way. After the cortex on each side had been thoroughly explored (*vide* infra) the cord was rapidly exposed (the animal being deeply etherised), and then being still living, the contrast between the dead white of the degenerated ascending posterior column which had been divided, as compared with the almost semi-translucent appearance of the normal one, was very striking.

The cord was then removed and hardened in a 2 per cent. solution of bichromate of ammonia, to which $\frac{1}{3}$ volume of methylated spirit was added at the end of a fortnight. Two portions of the cord were then taken, viz. (1) the seat of operation with the neighbouring 6mm. above and below the point cut, and (2) a small piece opposite the 1st or 2nd cervical nerve; these were soaked in water, then in gum, and finally cut after freezing. One half of the sections were then stained with aniline blue-black, the other with logwood, and finally mounted in oil of cloves and Canada balsam. This method of preparing the sections, although rapid, had the disadvantage of allowing the more friable grey matter of the posterior cornu, forming, as it necessarily does, the outer boundary of the wound in the spinal-cord to partly break away in some cases during the preparation of some of the sections, as is represented in the accompanying outlines of the various sections. (See pp. 56, 57.) The exact delimitation of the lesion to the posterior column was very obvious during the cutting of the preparations in the microtome. The condition of the wound in each case was closely similar, so that one description will serve for all. It consisted simply of a microscopic slit extending from the surface of the cord to the posterior commissure, and containing a little wound-serum and debris of damaged nerve-fibre, the mouth of the wound being always closed by young but firm connective tissue. The appearance of the sides of the wound-cavity, although so extremely small, showed clearly that there was some degree of tension in it; tension therefore was present in the interior of the cord, a factor that seems to me of some pathological importance in the correct estimation of the effects of such wounds of the cord. The chief point, however, which calls for particular notice in this method of research is the degeneration in some of the parts of the cord contiguous to the seat of operation, and of which a great deal has already been said. We may conveniently discuss this important point under the headings *Nature, Locality,* and *Causation.* The part which it plays in producing Prof. Schiff's result I have already referred to at full length, therefore now only wish to draw attention to the details of its existence.

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[1] Not wishing to stretch my point beyond what some might consider justifiable limits, I have purposely called the ascending fibres, described by Miescher as of "so-called vaso-motor influence," but it will be remembered that they were channels of conduction for afferent impressions produced by stimulating the great sciatic nerve

(1.) *Nature.*—Prof. Schiff, it will be remembered, insisted on an interval of a few days being allowed to elapse, in order, as he said, that the degeneration might reach the cortex. Now how far the ascending degeneration of the posterior columns gets in four or five days, I do not know, not having traced the course of the degenerated fibres; but this much is perfectly evident, that it does not arrive at, and cause the destruction of, the ganglion-corpuscles in the sigmoid gyrus, as Prof. Schiff would have us believe, for, microscopically, these corpuscles show no degenerative changes. But it is plain that this interval allows of the degeneration in the motor tracts of the spinal cord about to be described. Now the nature of this degeneration is very simple, consisting, as it does, merely in the swelling of axis-cylinders and myelin sheaths, so as to form the well-known and classical picture of the histological changes produced in nerve-fibres by acute "inflammatory" mischief within that space of time. In the accompanying drawings, where the shading has been marked darkly, there it is to be understood that the degeneration was very complete, that is to say, that all the nerve-fibres in the degenerated area were destroyed. To prevent misapprehension on this point, it will be well to draw attention to the fact that this complete destruction was only to be found in the direct cerebellar tract, the crossed lateral tract, and the direct pyramidal tract of the same side as the wound in the cord. I mention this particularly, since in Nos. 2 and 5 it will be noticed that the degeneration is necessarily represented diagrammatically as being spread over the whole lateral column, because degenerated fibres could be detected throughout that half of the section, although complete destruction was limited to the tracts above noted. It is scarcely necessary to describe the histological details at length, since, as has been stated above, they presented the ordinary appearances. It will make, however, this account more complete if I briefly state the chief facts:—

Changes in—

(A.) *Nerve Fibres.—Axis Cylinders.* The axis cylinders of the fibres involved in the degenerative process remain unaltered at first, since they can be detected in the centre of the swollen myelin (*vide* infra).

Soon, however, they atrophy, and in the majority of instances the space (dilated) in the neuroglia formerly occupied by the fibre is simply filled with a deeply staining myelin globule. The continuity of the fibre is thus completely interrupted.—*Myelin.* The myelin exhibits the first effects of the damaging process. It swells, and at first is but feebly stained by aniline blue-black; then it swells more, so as to fill the space in the neuroglia, and it stains nearly black. Finally, it appears to shrink again, and is absorbed or, as a rule, simply drops out in the preparation of the section.

(B.) *Neuroglia.*—The changes in the neuroglia, as might be expected, are not very marked, owing to the shortness of the period of time during which they might develop. In fact, as regards the fine supporting trabeculæ of connective tissue running with the capillaries, the only notable point about them is that they take the staining more deeply on the degenerated areas. The fine meshwork between them appears unaltered, save by the distension of the fibre spaces before referred to. As is noted in the description of each of the spinal cords at the seat of operation, the grey cornua in the neighbourhood of the wound are in most cases infiltrated to a greater or less extent with leucocytes, and there is also under these circumstances a distinct appearance of œdema.

(C.) *Blood-Vessels.*—The degenerated area in every case was very anæmic, and in one instance, No. 4, this was extremely marked, while in the right half of the cord the vessels were full of blood. The importance of this fact will be discussed directly. (*vide Causation*, infra.)

(2.) *Locality.*—The exact locality of the degeneration is shown in the accompanying sketches of the cords, *vide* pp. 56, 57, but attention may be drawn with advantage to one or two cardinal facts determining its distribution.

Thus at the first glance it will be seen that the mischief *is almost confined to the side of the cord operated upon.* Next, that it is most marked in the closely adjoining posterior third

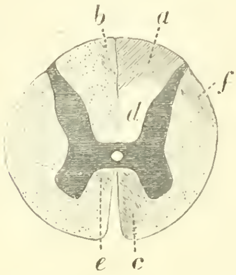

Fig. 1.—*Central Canal* distended, and contained exudation and blood. *The left posterior cornu* was infiltrated with leucocytes for half its breadth, and in some sections through its whole thickness.

Septic Case.—Pia mater was thickly infiltrated with leucocytes (pus). Schiff's result, marked.

a = Left posterior column destroyed, *i.e.* track of knife. *b* = Small portion of right p. c. degenerated. *c* = Whole direct pyramidal tract of left anterior column very greatly degenerated. *d* = Cavity of wound. *e* = Slight degeneration of direct pyramidal tract of right anterior column. *f* = Degeneration of ascending direct cerebellar tract.

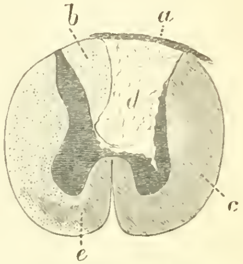

Fig. 2.—*Central Canal* was normal. It is not represented in the figure, which is a copy of the microscopical preparation. *The left cornua* were much infiltrated with leucocytes, and here and there patches of extravasated blood.

Aseptic Case.—Schiff's result, marked.

a = Scar tissue closing wound. *b* = Right posterior column (⅔rds.). *c* = Area of degeneration on left side involving whole antero-lateral column. *d* = Wound cavity full of debris. (*Note.* The damage done appears much larger, as already explained, owing to the friability of the "inflamed" grey substance.) *e* = Degeneration in the direct pyramidal tract of the right anterior column.

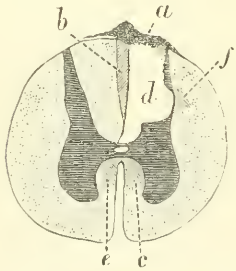

Fig. 3.—*Central Canal* contains blood clot.

Aseptic Case.—Schiff's result, very slight.

a = Scar tissue plugging wound. *b* = Remains of left posterior column destroyed. *c* = Degeneration, very slight in left anterior column. *d* = Wound cavity. *e* = Degeneration of right anterior column, slight. *f* = Slight œdema (?) of crossed lateral tract.

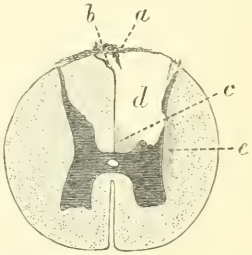

Fig. 4.—*Central Canal* moderately distended with exudation. *The left posterior cornu* was infiltrated with leucocytes at the notch represented in the drawing.

Aseptic Case. Schiff's result, extremely slight.

a = Scar tissue closing wound. *b* = Small angle of right posterior column cut away. *c* = Small patch of degenerated left posterior column. *d* = Cavity of wound. *e* = Small vertical patch of degeneration just outside cornu of grey matter.

of the lateral column. Thirdly, that the next place of greatest frequency is the direct pyramidal tract in the vertical line of the wound. The explanation of the greater liability of these areas to be affected will be discussed directly, *vide Causation, infra*; but I must now state that the degeneration is only marked exactly opposite the wound in the cord, and only extends a millimetre above and perhaps two millimetres below that point. This narrow limitation of the degeneration

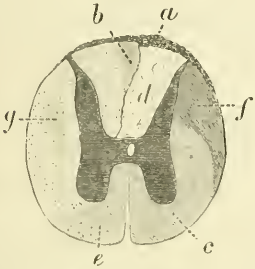

Fig. 5.—*Central Canal* normal. Whole cord, but especially left grey cornu, is infiltrated slightly with leucocytes. Septic Case. — Cord œdematous. Schiff's result, excessively marked, see p. 6.

a = Scar tissue and pia mater. *b* = Damaged portion of right posterior column. *c* = Degenerated left antero-lateral column. *d* = Cavity of wound filled with debris of left posterior column. *e* = Degenerated direct pyramidal tract in right anterior column. *f* = Degeneration of crossed lateral tract in left half of cord. *g* = Very slight degeneration in right crossed lateral tract.

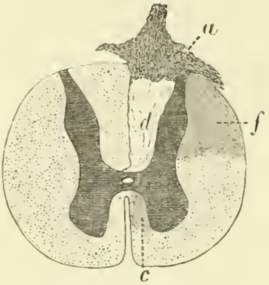

Fig. 6.—*Central Canal* normal. Left cornu infiltrated with leucocytes. Aseptic Case. Schiff's result, marked.

a = Scar tissue. *d* = Cavity of wound filled with debris. *c* = Degeneration in direct pyramidal tract of left anterior column. *f* = Crossed lateral tract and cerebellar tract much degenerated to exact limit shown.

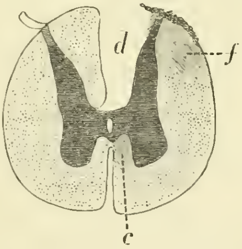

Fig. 7.—*Central Canal* normal. Aseptic Case. Schiff's result, very slight.

d = Wound cavity. *c* = Degeneration in left anterior column. (This was extremely limited.) *f* = Well-marked degeneration of the cerebellar tract, extending but slightly into left crossed lateral tract.

proves it to be an entirely local effect of the operation. I mention this, because it might possibly be imagined by some that ascending degeneration might, as Schiff believes, go up to the sigmoid gyrus, destroy the corpuscles there, and then come down the pyramidal tract. In each case I have examined the cord microscopically, not only just above and below the seat of operation, but also opposite the 1st and 2nd cervical nerves, and in no instance was any degeneration observable in the motor tracts at the summit of the cord. We may now discuss the causation of this degeneration.

(3.) *Causation.*—In the first place it is to be remembered that, with the exception of Nos. I. and V., each experiment was absolutely aseptic, and therefore we may set aside sepsis as a cause of the degeneration. From a consideration of the facts that the degeneration was most marked in proportion to

1. The amount of distension of the wound-cavity, in the cord and this means also the density and size of the cicatricial plug closing it;

2. The amount of anæmia and damage to blood-vessels; it is clear that we have suggested to us two ways in which this overlooked source of fallacy may be caused. These two possible ways are, tension in the cord, and interference with the blood-supply. In the first place, tension will obviously tell most heavily on the neighbouring crossed lateral tract. In the second place, the whole vascular supply to the left posterior quadrant of the cord will be interfered with by the operation, and further than that, a certain amount of thrombosis may occur which would reach the median artery, and so cut off the supply to the direct pyramidal tract. It would not be profitable to go over again the evidence offered in the foregoing pages, proving that both of these conditions follow the division of the posterior column, but it is worth while to note, that, as expressed also in the Table on p. 62, where these conditions were only slightly present the results as described by Schiff were very feebly marked, and vice versâ.

Having now seen clearly that a little localised patch of acute degeneration (plus tension in the cord) attacks invariably, though in very different degree, the motor tracts in the spinal cord on the same side as a wound of the posterior column, and that such a patch and such pressure necessarily form a block, offering greater or less resistance to the passage of motor impulse from the excitable cortex, we may turn to review the results obtained by stimulating the cortex in these experiments, as directed by Prof. Schiff. It will be remembered that in every case the *left* posterior column was the one divided. In the first two experiments I first exposed and tested the right excitable area, and then subsequently (*i.e.* ten or fifteen minutes later) the left. In the remaining five experiments I first removed the bone from the dura mater over both the motor cortices, and then raised the dura mater so that the exposure of the two sides was practically simultaneous, while in testing, the electrodes were first tried on one side and then on the other. This slight difference in the mode of treating the cortex on the two sides, as might be expected, made no difference in the results obtained. As will be seen from the subjoined Table, the amount of movement of the fore-limb of the left side as compared with that of the right side, when the right and left sigmoid gyri were respectively stimulated, varied inversely as the amount of degeneration visible in the motor tract of the cord opposite the seat of operation. Consequently it is erroneous to describe the diminution of movement as being due to a decrease in the excitability of the sigmoid gyrus, when it is really owing to the fact, that of course the swollen axis-cylinders in the degenerated lateral columns are incapable of transmitting the energy discharged from the irritated cortex. This experimental result is an interesting illustration of the clinical observation of paralysis from acute "inflammatory" conditions in the spinal cord. It will not be necessary to take up space with the minute details of all the cases experimented upon, but I may give short notes of Experiment V. as illustrating, first, the most extreme condition of Prof. Schiff's result; secondly, the fact that (as I have mentioned before) this result was obtained to such a very marked degree because, owing to the wound becoming septic, the degeneration was more extensive; and finally, the general method followed. The following is an abstract of the details of this experiment.

Experiment V.—Dog, Adult Mongrel Terrier.

Sept. 11*th*, 1885.—Left posterior column divided (according to method described above). Animal asphyxiated by ether, but recovered by artificial respiration.

12*th*.—Dog apparently well.

13*th*.—Marked paralysis of left fore-paw. Lies habitually on left side.

In attempting to walk, frequently falls to left side (hemiplegic).

14*th*.—During previous night, symptoms of spinal meningitis noted.

At 2.20 P.M. both fore-legs rigid, general rigidity of trunk, &c., with the other classical symptoms of meningitis. Dog immediately anæsthetised with ether.

2.45 P.M.—Both sigmoid gyri exposed within two minutes, and then stimulated as above described.

(I may here take this opportunity of stating, that in this series of experiments the apparatus of stimulation were an ordinary Du Bois Reymond induction coil, one Daniell cell [in one case, two cells were used] and usual platinum electrodes. The divisions indicating the distance of the secondary from the primary coil are given in centimetres; the strongest current, therefore, being at zero.)

Current.	Right Sig. Gyrus.	Left Sig. Gyrus.
10 cm.	Nil.	Nil.
8 cm.	Nil.	Nil.
6 cm.	Flickerings in left trapezius.	Fibrillar twitchings in right trapezius and protrusion of limb with extension of fore-paw.
5½ cm.	Ditto.	Ditto, stronger.
Interval, *i.e.* rest of 5 minutes.		
5½ cm.	Ditto, no spread.	Ditto, and spread to head, and right lower limb.

"The difference was so extremely marked as not to need further investigation."

In fact a superficial examination would have led one to state that the right sigmoid gyrus, as far as the centres for the limb- and trunk-muscles respectively were concerned, was inexcitable. As a matter of fact however, in this, as in all other experiments, the right cortex was perfectly excitable, as evidenced by the active movements of the facial and cervical muscles as low as the distribution of the fourth cervical nerve. I have already described the condition of the spinal cord in this case (see page 57), in which description it will be remembered that the degeneration of the motor tract was most marked. This case will thus serve as a typical illustration of the mode in which I believe Prof. Schiff's result is always produced. Of course it was impossible in this instance that the nerve energy should reach the left limb muscles.

The rest of the facts concerning the production of Schiff's result, viz. the diminution in the usual amount of muscular movement evoked by stimulation of the opposite sigmoid gyrus, so invariably illustrate (*vide* Table, p. 62) the fact alluded to before, viz. the variation of degree of loss of power, being proportionate to the damage of the lateral columns, that but little addition is necessary to the details just enumerated. There are, however, one or two points which are of considerable interest. In the first place, in those cases where Schiff's result was scarcely obtained at all it was only obvious when powerful currents were employed. Moderate, but very adequate stimuli called forth the usual movements on both sides, if a little different in force, but with such a stimulus as a current at 5 cm. this difference in violence of the movement became very apparent. The second point is this, that in three instances there was noted very definite clonic tremor (post-ethereal) of a rate of ten to twelve per second, which occurred in the left fore-limb first before invading the others. This fact seems to me worthy of note, since it appears to indicate that ordinary clonic tremor, so familiar clinically, is, as I suggested in my Brown Lectures, 1885, not the simple question of a rhythmical alternation of contraction of opposing groups of muscles, but that it (ankle-clonus, for example) is really the rhythmical contraction of a given muscle (*e.g.* the gastrocnemius) in response to a rhythmical discharge of the spinal motor corpuscles governing that muscle, such corpuscles having been thrown into discharging activity by the sudden violent afferent impulse which is generated (according to the usual clinical method) by forcibly stretching that muscle, *e.g.* by flexion of the ankle. The pathological irritability of the motor corpuscles in my experiments obviously is set up by the changes I have described in the descending pyramidal tracts, an experimental condition comparable to the pathological one of sclerosis.

Conclusion.—The inference to be drawn from the above experimental examination of the question whether Schiff's method shows the posterior columns to ascend directly to the motor or excitable cortex must, I think, be that such direct connection is *not proven.* The investigation (the expenses of which were defrayed by a grant of 5*l.* from the British Medical Association) has elicited the fact, not, so far as I know, published before, that tension and anæmia in the cord will bring about marked local degeneration in the motor tract especially.

TABLE showing the relation between the DEGENERATION in the ANTEROLATERAL COLUMNS and the EFFECTS which were supposed to be due to LESION of the POSTERIOR COLUMN, this being in every case completely divided.

No. of Experiment.	Paresis.	Tactile Anæsthesia.	Schiff's result of stimulating opposite excitable area.	Degeneration in Motor tract.	Remarks.
I.	Present.	Could not be tested.	Marked.	Marked.	Septic.
II.	Present.	Present.	Marked	Marked.	Aseptic.
III.	Absent.	Absent.	Very slight.	Very slight.	..
IV.	Absent.	Absent.	Very slight.	Extremely slight.	..
V.	Almost Hemiplegic.	Could not be tested.	Very marked.	Extreme.	Septic.
VI.	Present.	Present.	Marked.	Marked.	Aseptic.
VII.	Absent.	Absent.	Very slight.	Slight.	..

NOTE.—When Schiff's result of want of movement following stimulation of the right sigmoid gyrus as compared with that following excitation of the left is set down in varying degree in the 4th column, it is to be remembered that the terms in which it is described are exact, since they are founded on the experimental data which are the measurements of the strength of the current in centimetres of the distance of the secondary from the primary coil; see p. 60.

ON PROFESSOR HAMILTON'S THEORY CONCERNING THE CORPUS CALLOSUM.

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In the last number but one of 'Brain' (October, 1885), a short preliminary note was given by me in reference to the view propounded by Prof. Hamilton, that the corpus callosum is not an interhemispheral commissure at all, but is the decussation of the cortical fibres in their progress downwards to become connected with the basal ganglia.

It is proposed in this paper to test the theory by the evidence afforded by the microscopical examination of very thin sections stained by Weigert's hæmatoxylin method, and by comparing the corpus callosum in man with that of the other mammalia, and especially with regard to the relative size between the anterior commissure and the corpus callosum in the lower animals.

I will at once begin with the evidence which microscopical examination gives us on this question.

With regard to the evidence of microscopical examination, it should be stated that my sections have been made chiefly from the monkey; for although many sections have been made from the brain of man, I have relied especially on those of the former, for the reason that the brains of these animals can be had absolutely fresh, and can be put into the hardening fluid directly after the animal is killed. Besides the serious difficulty of obtaining the brain of man fresh, it is too large to trace with accuracy the fibres to any distance, when viewed under the higher powers of the microscope. It would be well to say at once that I have not in any way relied on the naked-eye appearances, or the mere use of a magnifying glass, but all the sections have been examined under various powers of the microscope. The brain of the marmoset has also been examined, and has proved very easy of manipulation, owing to its small size.

The brains taken from the monkey were treated as follows: the animal was killed with chloroform, and at once the brain was removed and put whole into a mixture of methylated alcohol and water (half and half) for 24 hours, then it was cut into pieces of $\frac{1}{4}$ inch thick—frontally, horizontally or sagitally, as the case might be—and put into bichromate of potash solution 3 per cent., for three to four weeks, and afterwards the hardening was completed by methylated alcohol. The pieces were imbedded in collodion and sections cut; they were then stained by Weigert's hæmatoxylin method, and passed through methylated alcohol, absolute alcohol, oil of cloves, and mounted in Canada balsam.[1] The marmoset's brain was stained in toto by Weigert's hæmatoxylin, and cut in paraffin, as described in 'BRAIN,' July, 1885.

In some cases the brains were sliced and put at once into bichromate-of-potash solution. On looking at a section cut from a brain prepared in the above way, the individual medullated fibres will be seen stained quite black, and their course can thus be well made out.

If we examine a frontal section taken from about the posterior part of the corpus callosum (Fig. 1) on a level with the middle of the pons Varolii, it will be seen that the corpus callosum (C C) on reaching the hemisphere on either side spreads out like a fan, sending fibres upwards to the convolutions at the vertex (C′ C′) and outwards to all the convolutions down to a certain level. Now this level is found in the monkey to be the upper part of the Island of Reil; I have examined most carefully with the high power of F Zeiss, but I have failed to find any transverse or oblique fibres of the corpus callosum below this point, they seem to stop suddenly. This fact is therefore at variance with Prof. Hamilton's view, who considers that many of the fibres of the crossed callosal tract end in the external capsule.

With regard to the internal capsule, I cannot find a single fibre which passes from the corpus callosum into the internal capsule; the lowest fibres of the corpus callosum run horizontally outwards to the upper part of the Island of Reil, at right angles to the fibres issuing from the external and internal capsules; the corpus callosum does not pass into either of the capsules, in the monkey or in man.

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[1] 'Fortschritte der Medicin,' 1885, No. 8.

The fibres issuing from the internal capsule (C I) course upwards, at first at right angles to the callosal fibres, but higher up they cross them obliquely (C′I′), and can be traced the whole way from the capsule to the grey matter of the convolutions of the same side. The most internal fibres from the internal capsule have a more curved course than the more external ones; for three-fourths of their course across the callosal fibres they are easy to trace, but after this point it is more difficult to follow them; but by comparing different sections and using high powers they appear to go to the lower part of the marginal convolution; in my opinion they certainly do *not* go into the corpus callosum; these fibres are the only ones which present any difficulty in ascertaining their destination, as all the other fibres from the internal capsule can be easily traced to the convolutions of the same side.

From the external capsule (C E) only a few fibres come from the part internal to the claustrum and ascend with the fibres from the internal capsule on their way to the convolutions of the same side.

These fibres from the external capsule are situated externally to the main mass coming from the internal, but they are crossed by fibres (O P) passing from the internal capsule to the convolutions on the upper border of the Sylvian fissure. The fibres from the external capsule do not, in my opinion, come into any relation with the corpus callosum except to cross at right angles to its lower fibres, and to run parallel with its upper fibres on their way to the vertex; the two are certainly not continuous.

The brain of the marmoset presents very important evidence in favour of the commissural theory of the corpus callosum. I have managed to stain this brain in toto by Weigert's hæmatoxylin,[1] and have cut the whole brain frontally into sections, from the optic chiasma in front to the posterior part of the pons behind; every section has been looked at, and only those have been mounted in series which present any naked-eye difference to those preceding them; by this means we get a series of over two dozen sections showing all gradations of change, between the optic chiasma and the hinder part of the pons. On examining all these sections with the highest powers of the microscope, I have failed to find a single case where the fibres of the corpus callosum pass into the internal capsule. As has been mentioned in the preliminary note,[1] the brain of the marmoset differs from that of the other apes in the fact, that the gyrus fornicatus does not exist as a separate convolution, lying on—or appearing to spring up from—the upper surface of the corpus callosum, but it is more blended with the convolutions at the margin of the longitudinal fissure, so that in a frontal section the medullated antero-posterior fibres of the gyrus fornicatus (cingulum) form a projection at the lower part of the inner face of the centrum ovale above, and to the inner side of the peripheral end of the corpus callosum. This simplifies matters very much, and we do not have any fibres from the internal capsule making their way up through the corpus callosum before it begins to spread out.

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[1] 'BRAIN,' July, 1885.

This disposition of the gyrus fornicatus in the marmoset, and the fact, alluded to further on—that the fibres of the internal capsule have a course which is directed almost quite straight upwards and inwards—have, I think, a most important bearing in understanding the relations of the corpus callosum and the internal capsule. I believe that the large curve which the fibres of the internal capsule in man make, with the concavity of the bend towards the corpus callosum, has led Prof. Hamilton to suppose that the internal capsule actually passes into the corpus callosum. He has shown these curved fibres in his figure in the 'Journal of Anatomy and Physiology,' but he has not drawn them actually passing into the corpus callosum. In the monkey these fibres from the internal capsule are less curved; while when we come to the marmoset we find them almost straight, and at right angles to the horizontal axis of the corpus callosum.

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[1] 'Brain,' October, 1885.

In the marmoset the arrangement in all these sections is this: the corpus callosum spreads out its fibres like a fan before reaching the centrum ovale, and sends the upper part of this fan upwards, outwards, and then inwards to just above the gyrus fornicatus; the middle part through the internal capsular fibres to the cortex, while the lower part runs downwards and outwards to the cortex, meeting and crossing the fibres coming up from the internal capsule, and here a well-marked decussation occurs between these different sets of fibres.

The fibres from the internal capsule have nearly a straight course upwards and inwards to reach the convolutions at the vertex of the same side; they do not present the curve, concave inwards, which is seen in the other apes and man, and in no place do they pass into the corpus callosum, nor does this latter send any fibres into the internal capsule.

Prof. Hamilton has stated that the pyramidal tract, alone of the fibres forming the corona radiata, does not pass into the corpus callosum to decussate to the opposite side, but that the rest of the fibres, anterior and posterior to the pyramidal tract do pass into the corpus callosum. Now if this were the case we should find at the borders of the pyramidal tract a sudden change in the disposition of the fibres coming up from the external and internal capsules, they would form a sharp line of demarcation on each side of the pyramidal tract which would show where the crossed callosal tract begins. With regard to the corpus callosum itself, at the level of the pyramidal tracts (in frontal sections), what is its function there? It cannot be a decussation, as we are distinctly told that the pyramidal tracts are alone exempt from the callosal decussation,[1] and it is not a commissure between the hemispheres, it surely ought to be absent altogether at this point, and we should have a blank space opposite the pyramidal tracts. I must maintain, from an examination of my marmoset sections which were made in front of the optic commissure, as well as from those between this point and the hinder part of the pons, that there is absolutely no difference in the relation of the corpus callosum to the capsular fibres; there is no change of structure at the anterior or posterior margins of the pyramidal tracts different to what is seen in a section made through the centre of these tracts.

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[1] 'Liverpool-Medico-Chirurgical Journal,' Jan. 1886.

To show the arrangement of the corpus callosum and the internal capsule in the anterior part of the brain, I have cut horizontal sections of the brains of man, the monkey and marmoset, prepared in the way mentioned above, and stained with Weigert's hæmatoxylin. The relation of the parts is seen in Fig. 2, which is a horizontal section through the left genu of the corpus callosum and the anterior fibres of the internal capsule. The under part of the genu of the corpus callosum is here seen to be divided into two parts, an anterior and posterior; the anterior part (C′ C′) begins in the extreme tip of the frontal convolutions, and runs backwards and then inwards to cross over to the corresponding part on the opposite side, forming a true commissure between the two frontal convolutions; the posterior part (C C), quite distinct and separated off from the anterior, passes from the middle line of the corpus callosum, and runs outwards till it meets with the fibres coming from the internal capsule (C I). This latter has its fibres in parallel bundles until it has passed the lenticular and caudate nuclei (N L N C), it then reaches the medullary white matter, and sends its fibres anteriorly, externally, and posteriorly. The anterior fibres (C′ I′) go to the tip of the frontal convolutions and form a very sharply defined bundle, which can be traced the whole way into the frontal convolutions of the same side; the whole of these anterior fibres can be seen passing through the fibres from the posterior part of the germ of the corpus callosum (C C), with which they make a sharp angle and a well-marked decussation (D); while the posterior fibres of the corpus callosum make their way to the cortex in front of the fissure of Sylvius.

It is quite evident that the anterior fibres go straight from the internal capsule to the tip of the frontal convolutions, and in no case can I find the fibres of the corpus callosum passing into the internal capsule; the two parts of the corpus callosum are quite distinct, and no decussation in the middle line takes place between them.

The decussation of the corpus callosum with the internal capsule, which is so evident at the angle where these fibres of the internal capsule and corpus callosum first meet, can also be seen by a high power to take place in the mass of white medullary substance in front of, and opposite to the point of entrance of the internal capsule.

From comparative anatomy there are several important facts which throw much light on the structure of the corpus callosum, and this is especially the case when comparison between the relative size of the corpus callosum and the anterior commissure is made in different animals.

In man the diminutive size of the anterior commissure as compared with the corpus callosum is well known; but already in the monkey it can be seen in a frontal section that the vertical cross area of the anterior commissure is almost as large as that of the corpus callosum, although of course the longitudinal area of the latter is many times in excess of the former. This relation between the two commissures was pointed out by Prof. Flower in a most interesting and valuable paper,[1] where he calls attention to the excessive development of the anterior commissure, and the small size of the corpus callosum in the marsupia. Prof. Flower's paper is illustrated with drawings of the frontal and sagittal position of brains of various animals, from man down to the marsupia and monotremata.

I have lately, through the kindness of Mr. Beddard, prosector at the Zoological Gardens, obtained a brain of the large kangaroo (*Macropus major*); this has been hardened and stained by the above methods, and frontal sections have been cut through the corpus callosum and the anterior commissure, so as to show both at once. In this animal, as described by Prof. Flower, the corpus callosum is very small indeed, especially when seen frontally, and is a mere thread—so small that Prof. Owen hardly considered it to be a corpus callosum; but on this point I venture to think that there can be no doubt that Prof. Flower's opinion is correct. When this brain is stained by Weigert's method, the corpus callosum is seen to pass from one gyrus fornicatus to the other, and to act as a commissure between these convolutions only. On the other hand, the anterior commissure is enormously developed, and occupies one-fourth of the whole vertical height of the brain;

[1] 'Philosoph. Transact.,' 1865, Pl. xxxvii. and xxxviii.

its fibres form a thick band in the middle line, and on each side it divides into two bands which run into the hemispheres on either side, and acts as a commissure between the upper convolutions, as well as between the lower of either side. The bands of fibres can be traced completely across from side to side without decussating.

The difference of the size between these two commissures in different animals seems evidently due to the difference in the size of the parts of the brain they have to join; in the kangaroo, the temporo-sphenoidal convolutions are enormously developed, while the frontal region is very small, and hence the small size of the corpus callosum as compared to the anterior commissure. This condition in the kangaroo has a very important bearing on the structure and function of the corpus callosum in man and the higher mammalia. In the kangaroo, it is perfectly impossible that the thin shred of a corpus callosum can be the path of decussion of the crossed callosal tract, coming from the internal capsule; besides which, the fibres of the corpus callosum can be seen to end in the gyri fornicati. It is evident that the only path left for the crossed callosal tract must be the anterior commissure; but this is in front of the pyramidal tract, and the anterior fibres of the corona radiata are here seen to be cut transversely, and to have no connection with the anterior commissure.

This point alone seems to me to be very strongly against Prof. Hamilton's theory of the corpus callosum.

He has asked the question, How do animals which have no corpus callosum manage to perform combined movements of the two sides, and he considers that the absence of this body militates against the theory that it is a commissure between the two hemispheres. I think this question is sufficiently answered by the fact, that as we descend the mammalian scale, the anterior commissure is developed in proportion as the corpus callosum diminishes, until in the marsupialia the former attains the enormous size seen in the kangaroo.

In the kangaroo neither the corpus callosum nor the anterior commissure can be considered as a crossed callosal tract, and it cannot, I think, be conceived that in the lower mammalia the brain should be formed on a type totally different to that of the higher animals; this must be contrary to all ideas of evolution.

In the 'Liverpool Medico-Chirurgical Journal' for last January, Prof. Hamilton has done me the honour of referring to my preliminary note in 'BRAIN,' October, 1885. He there complains that I have not followed a single one of the methods he employs, and that the clarifying process I use is so coarse as in itself to destroy any chance of getting a view of the continuity of the crossed callosal tract, and that I shrink all the beauty out of the callosal fibres by employing spirit as a hardening reagent.

The method which I use—Weigert's hæmatoxylin—has the property of staining all the medullated fibres of the brain, and is so delicate and perfect as to show completely the finest fibres of the cortex, and present such a delicate network there, that they can only be fully appreciated by using high powers, as the F Zeiss. And we are to suppose that these minute fibres are to be everywhere stained in my preparations, and yet the crossed callosal fibres of the bulky corpus callosum—which Prof. Hamilton can see with an ordinary magnifying glass—are in some mysterious manner shrunk away by using methylated alcohol, after hardening in bichromate of potash for a month, and by my coarse clarifying agent,—oil of cloves! With regard to this latter reagent, I can positively state that no disappearance of fibres occurs, as I examine the sections under a low power first in water, then in alcohol, and finally in the oil of cloves; there is only this difference, viz. that in the oil of cloves the details are more clearly brought out, owing to its well-known properties of clearing the specimen. Further comment is, I think, unnecessary.

I have not employed Prof. Hamilton's method; as I consider that swelling and separating the fibres by the action of potash and gelatine must tend to disturb their normal arrangement much more than alcohol or oil of cloves, and I should much like to know whether by his method he is able to show the finest plexus of fibres in the cortex, or, if they are visible, whether they are seen as clearly as they are by Weigert's hæmatoxylin method; until that is possible I shall continue to employ the latter method.

Although I am charged with "shrinking all the beauty out of the callosal fibres by employing spirit as a hardening reaction," what do we find in the description of Prof. Hamilton's process in 'BRAIN,' July, 1883, p. 214? We are actually told there to put the pieces of brain into pure methylated spirit, to finish the hardening after Müller's fluid,—precisely what I have done with my preparations. The use of Weigert's säure fuchsin has been alluded to in some of Prof. Hamilton's papers, but as he soaks his preparations for four days in water, and then in an aqueous solution of mucilage and syrup for two weeks or more, it is quite impossible that he has obtained the proper reaction. I make this statement with due caution, as, having had the advantage of working with Prof. Weigert at the time he made this discovery, and being, I believe, the first to put it to practical use in my work on the cerebellar cortex at the Physiological Laboratory at Leipzig, where I used this dye daily for six months, I can endorse the canon that Prof. Weigert has laid down, viz. that on no account may the pieces of tissue be put into water before using the säure fuchsin.

Prof. Hamilton states that I have not employed oblique sections as he directed, and yet the only two drawings which I have seen published—in the 'Journal of Anatomy and Physiology'—are perpendicular transverse sections; I have however made obliquely frontal sections of the monkey's brain, but have failed to see the crossed callosal tracts.

The two drawings, mentioned above, are drawn presumably the natural size of the human brain, and although the crossed callosal fibres are figured, they are not drawn (in Plate xxii.) as actually passing into the corpus callosum; they end abruptly, and might equally well pass on to the cortex of the same side.

Will Prof. Hamilton publish a *microscopical* drawing of these fibres? As in his papers, with the exception of that "on the Corpus Callosum in the Embryo," little allusion is made to the microscopical appearances, and in his last paper in the 'Liverpool Medico-Chirurgical Journal,' he states that he "cannot at present enter into the technicalities of the microscopic examination."

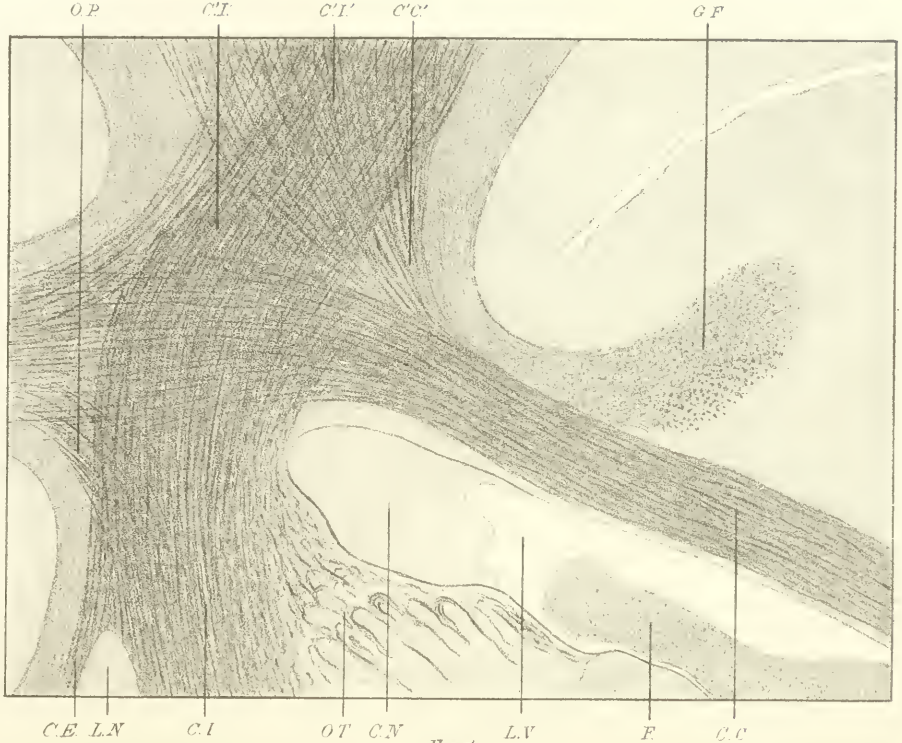


Fig 1

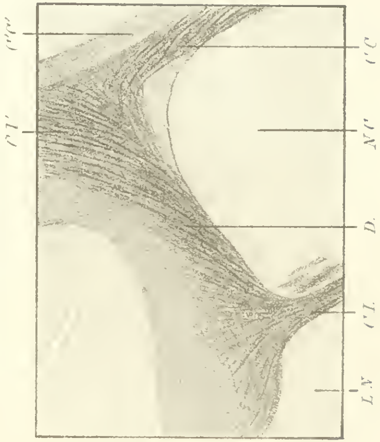


Fig 2

From a careful examination of this question from the point of view of anatomy and comparative anatomy, I can only come to the conclusion, that the generally received idea of the corpus callosum is quite correct.

The corpus callosum I consider to be a commissure between the two hemispheres; and the fibres of the internal and external capsules, in my opinion, do not pass into the corpus callosum to decussate to the opposite cortex; no distinct decussation can be demonstrated in the middle line of the corpus callosum, and the corona radiata does exist as such, and is not a nonentity, as Prof. Hamilton would wish us to consider.

EXPLANATION OF THE PLATE.

FIG. 1—is a *frontal* section through the left centrum ovale of a monkey's brain, and shows the decussation between the corpus callosum, and the internal and external capsules. The section is made at the vertical level of the middle of the pons Varolii, and through the hinder part of the corpus callosum, just in front of the splenium. The preparation was stained with Weigert's hæmatoxylin; and the drawing is in no way diagrammatic.

Magnified 10 diameters, and reduced.—C.C. left half of corpus callosum.

C'.C'. fibres of corpus callosum to convolutions at vertex.—C.I. Internal capsule.

C'.I'. fibres of internal capsule to convolutions at vertex, decussating with corpus callosum.

C.E. external capsule sending fibres to vertex, decussating with corpus callosum.

O.P. fibres from external and internal capsules to supra-marginal and ascending parietal convolutions.

L.N. lenticular nucleus.—C.N. caudate nucleus.—O.T. optic thalamus.

L.V. lateral ventricle of left side.—F. left half of fornix.—G.F. gyrus fornicatus.

FIG. 2—is a *horizontal* section through a portion of the anterior left half of a monkey's brain, and shows the decussation between the corpus callosum and the internal capsule. Prepared like Fig. 1.

Magnified 10 diameters, and reduced.

C.C. the left half of the genu of the corpus callosum, the hinder part of which turns backwards to decussate with the internal capsule.

C'.C'. the anterior part of the genu of the corpus callosum, which joins the anterior frontal lobes.

C.I. anterior fibres of the internal capsule.

C'.I'. fibres of internal capsule to anterior frontal lobes, after decussating with corpus callosum.

D. point of decussation of internal capsule and corpus callosum.

N.C. left caudate nucleus.—L.N. left lenticular nucleus.

Clinical Cases.

CASE OF INJURY TO SKULL.—TREPHINING FOR EPILEPTIFORM ATTACKS.—REMOVAL OF DEAD BONE.—DEATH.

BY RICHARD DAVY, M.B., F.R.S.E.,

Surgeon to the Westminster Hospital.

With a NOTE *on the* PHYSIOLOGICAL *and* MEDICAL ASPECTS *of the* CASE,

BY A. HUGHES BENNETT, M.D., F.R.C.P.

T. N., æt. 22, gasworker, single, was admitted into the Westminster Hospital on December 22, 1884, with a small scalp-wound, and injury to calvarium over the left parietal bone. Mr. Waller, the house surgeon, found that his probe entered the skull vertically for an inch downwards, and free bleeding was present. The patient stated that his head had been knocked by a falling iron chain; and that a hook at the end of the chain had swung, and caught him on the side of his head. His gait was unsteady and uncertain; he was quite conscious, and was at once placed quietly in bed. No signs of compression; a feeling of numbness of right side. He went on well, without any marked change, until Jan. 5, 1885. He then had marked twitchings of both arms and legs, followed by two epileptiform attacks; and on recovering, partial paralysis of his right face, right forearm, and right leg supervened; accompanied with a feeling of numbness of the right leg and arm. No paralysis of sphincters. My colleague, Mr. Thomas Bond, seriously discussed the propriety of trephining the skull over the wound; but postponed immediate operation.

Jan. 9th, 1885.—His urgent symptoms have subsided; so I restricted his diet to milk, one egg, bread and butter, and tea, and ordered open-air treatment to the wound, with cleanliness. I visited him daily during January, and with slight variations he continued to improve.

Feb. 2nd.—12.15 A.M. A marked epileptiform fit, followed by sickness. 3 A.M. Another, of 7 minutes' duration; cephalalgia, twitchings of both legs, especially the right. 7.30 A.M. Another fit, lasting 4 minutes. 11.22 A.M. Another fit, lasting 3 minutes.

At 1 P.M. I saw the patient in a fit, in consultation with my colleague Dr. Hughes Bennett; and we agreed to trephine over the wound at once. The man was chloroformed; a small exploratory trephine was used first at the edge of the original wound, and the circle of bone removed, and carefully examined on its dura-mater aspect, to see if the vitreous table had been broken by the accident. On finding this quite sound, a large trephine was used at the opposite part of the edge of the wound; on removal, this portion was found to be chipped irregularly, and marked the original injury; division of the dura mater permitted eight pieces of loose bone to be taken out of the brain substance; the deepest being two inches below the surface of the skull cap. My little finger carefully introduced through the trephine hole could feel the brain pulsating, but could detect no more detached fragments.

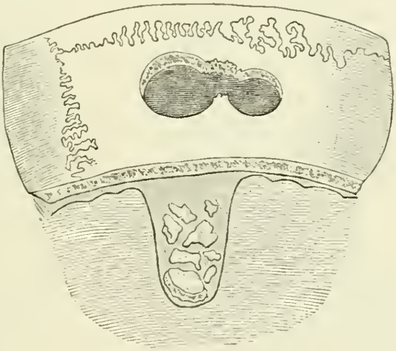


FIG. 1.

The diagram (Fig. 1) shows well the site of the original wound, trephine apertures, and the spiculæ. Some free hæmorrhage occurred, which was allowed to escape; for no dressings were used. The man slowly recovered from the chloroform, and had no fit until the next morning at 3 A.M. The patient was actually in a fit at the time of operating.

Feb. 3rd.—During the day had seven epileptiform attacks, each lasting a few minutes. These were preceded with marked twitching of the right arm; which the patient asked to be restrained. This day at 5 P.M. Dr. Hughes Bennett ordered him potass. bromid. gr. xxx. in water every six hours.

4th.—Good night, no fits; no marked headache. Pupils act equally. Right arm weak.

5th.—On account of diarrhœa the bromide of potassium was discontinued. Wound discharging; but its upper part has healed by first intention.

7th.—Pain in the right side of his chest and abdomen. Dry pleuritic rub. Muscular movement of his right arm has much improved.

8th.—Restless night; complains much of pain in his right arm and his chest. Posterior base of right lung is dull; dyspnœa; and friction-rub plainly to be heard. No headache.

10th.—Discharge from wound less; no fits. Restless from cough and pain at his right chest. States that he has had previous similar attacks.

12th.—He passed a good night; but his chest trouble is increased, crepitant râles heard all over his right chest; increase of vocal resonance, and frothy expectoration tinged with blood. His face is pallid; his lips blue. Tongue dry, brown and raspy. Pulse 126. Respirations 48.

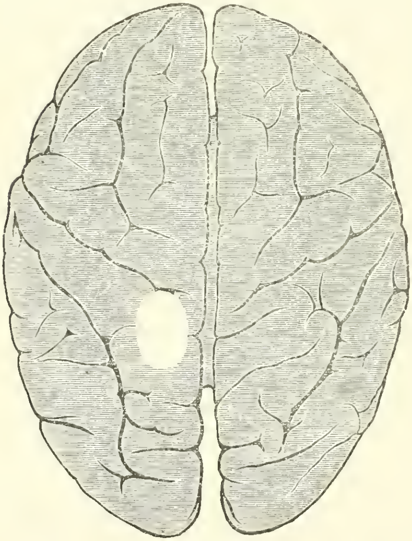


Fig. 2.

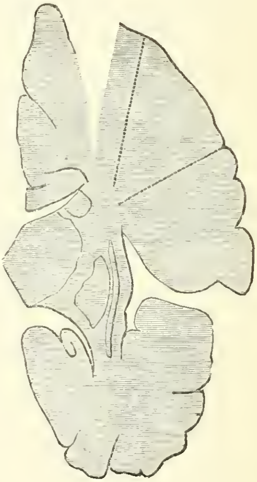


Fig. 3.

16th.—His symptoms became more marked; dyspnœa increased; and prostration intensified. He has not complained of any headache; his wound has given no trouble, and is entirely healed, except at one small point. He has not lost consciousness since February 3rd, 1885; and died, in a calm, rational, and exhausted condition at 12.10 A.M.

Post-mortem, February 18th.—An examination of the *head only* was allowed.

There was a scar on the left side of the vertex, which had recently healed, except at a point large enough to admit a small probe. This scar was situate over the left parietal bone, 7½ inches from the bridge of the nose, and about 1 inch to the left of the vertex. There was only a little discharge, no smell; and no hernix cerebri. The calvarium was carefully sawn off and removed. The brain surface was adherent around the trephine apertures by organised lymph, and tough fibrous tissue. Imbedded in this, and intimately adherent to the dura mater, was a shell of vitreous table, as large as a canary seed. There was no surrounding cerebritis, nor meningitis. The superior longitudinal sinus was severed; and its nasal end was plugged with a firm fibrinous decolorised clot. A ragged wound, large enough to admit a small finger, descended into the brain substance; this was situated at the upper part of the ascending parietal convolution, and immediately in front of and partly involving the paracentral lobule. There was no pus present in the brain, nor any spiculæ of bone. The exact site of this brain lesion is shown in the two plates.

There were marked evidences of mustard-poultices and of dry cuppings over both sides of the chest, and intercostal bulging with dulness, on percussion, over the right half of the chest; but no further examination of the body was allowed.

Remarks by Mr. R. Davy.—By this injury certain results were produced of a localised character. For fourteen days after the accident the patient was free from fits, and then within three days he had three marked epileptiform attacks. After an interval of twenty-five days the attacks recurred, and operative interference was therefore considered necessary.

The treatment of the wound by the open method appears to be satisfactory, no hernia cerebri resulted, and healing was evidently all but secured. The chest affection appeared to be repetition of an old attack; but unfortunately the denial of a complete post-mortem leaves this man's variety of pleurisy and pneumonia a doubtful point.

He died fourteen days after the operation; and in my own experience, chest complications (after injury to the brain) appear to be a common sequel.

I must express my obligation to Dr. Hughes Bennett for his assistance rendered in this trying case, and I have also requested him to add his own postscript to the preceding notes.

Note, by Dr. Hughes Bennett.

Mr. Davy having described the surgical aspect of this interesting case, has requested me to add a note from a physiological and medical point of view. Having had opportunities of watching the man during his illness, and believing that the case presents features of importance, at the risk of some repetition I give the details as they came under my notice, in so far as they bear upon the question of cerebral localisation.

I saw the patient for the first time on December 30th, that is, eight days after the accident. He was perfectly well as regards general health, and all his organs and functions were normal. Intelligence was intact. Special senses normal. No pain in head. Pupils equal and normal. Fundus oculi natural. No apparent asymmetry of face. The patient said the right arm felt numb, but, on the most careful testing, there was no trace of objective loss of cutaneous or muscular sensibility. The movements of the right hand and fingers were only slightly clumsy, but otherwise no definite impairment in the actions of the upper extremity could be detected. The tendon reflexes and irritability of the muscles to percussion were the same on both sides. The movements of the right lower extremity were distinctly weak. The patient could walk, but with some difficulty. When lying in bed, the various actions of the right leg were performed but slowly, weakly, and unsteadily; the left was normal. Cutaneous and muscular sensibility of both lower limbs was natural. The knee-jerk was increased on the right side, but not to a great extent. Well-marked ankle-clonus right side, imperfect left. The exact position of the wound on the scalp was three-quarters of an inch to the left of the longitudinal line, and one inch behind a vertical line drawn through the external meatus of the ear.

During the next month the patient remained in exactly the same condition, with the addition, that he was occasionally seized with epileptiform attacks, as referred to by Mr. Davy. According to the statements of the nurse, these were all of the same kind. One of them I personally observed. Suddenly the right leg became rigid and extended; this rapidly spread throughout the whole of the right side; the patient then apparently lost consciousness, and the entire body, including the face, entered into a state of clonic convulsion; which lasted for a few minutes. After the attack, the patient was drowsy for an hour or so, and then regained his normal condition.

From this series of facts it was concluded that there was a lesion of the cortex cerebri on the left side, causing irritation and partial destruction of the area presiding over the movements of the right lower, and to a lesser degree over the right upper extremities. This was evidenced by the permanent paresis of the right leg and the slight weakness of the right hand, and by the epileptiform convulsions beginning in the right lower extremity, extending throughout the side and ultimately over the entire body. From the position of the wound on the skull it was concluded that the wound of the brain was situated at the upper part of the ascending frontal convolution close to the longitudinal fissure. The diagnosis was confirmed in every respect by the operation, except that the injury to the grey matter was found further back than would have been anticipated from the position of the blow on the skull. The exact comparative size and locality of the destruction of brain substance caused by the manipulations necessary to remove the pieces of bone are indicated in the annexed diagrams. It will be seen that the cortex is absent throughout its entire thickness in a defined oval area at the upper part of the ascending parietal convolution, close to the longitudinal sulcus, and partly involving the postero-parietal lobule. Immediately below this a triangular portion of the corona radiata is destroyed. After death the parts marked white in the diagrams were strictly defined, occupied by firm cicatricial tissue, with normal cerebral matter in their immediate vicinity. The symptoms observed from the date of operation to the death of the patient from lung disease may be said to represent the effects caused by this local lesion of the brain. These were as follows: The patient was perfectly intelligent. The face and tongue were natural. The right upper extremity was slightly weaker than normal, but all its movements could be accurately performed. The right lower extremity was partially paralysed. It was not judged advisable to test locomotion; but when lying in bed, although the patient could flex and extend the limb, he did so slowly and feebly. It was difficult to say whether any special muscles were involved; there appeared to be general paresis of the whole limb. In the upper extremity the hand and fingers were chiefly affected, but even these only to a slight extent. There was nowhere any discoverable loss of sensibility. The tendon phenomena were increased in both lower extremities, chiefly on the right. The organs and functions of the body were otherwise normal.

The conclusion to be derived from the above observations is that a lesion of the cortex cerebri and subjacent corona radiata, strictly limited to the parts marked in the diagrams, is capable of causing marked but not complete paralysis of the lower, and slight paresis of the upper extremities of the opposite side, without any apparent disturbance of any other function or organ of the body.

A CASE OF CORTICAL PARALYSIS.

BY JOSEPH WIGLESWORTH, M.D. (LOND.)

Assistant Medical Officer, Rainhill Asylum.

ANN R., æt. 65, widow, was admitted into Rainhill Asylum, Sept. 8th, 1884, suffering from an attack of ordinary mania; this was her second attack, she having been under treatment in the same asylum for a similar mental affection, 5½ years previously. There was nothing at all peculiar in the character of the mental symptoms, which need not be again referred to. What concerns us here to note, is that she suffered from extensive necrosis of the skull, which was doubtless of syphilitic origin. At the time of her first admission, the inflammation of the cranial bones was actively in progress. We had on this, her second admission, to deal chiefly with the results of this disease, large portions of bone having come away, extensive irregularities of the upper surface of the cranium having thus been produced; over most of the depressions the skin was firmly cicatrised, but in some places small sores remained, covered with dry scabs.

About the middle of February, 1885, patient was laid up with a mild attack of facial erysipelas; on recovery from which (20th inst.) it was noticed that pulsation existed at the bottom of one of the cranial depressions. Though no bone was found, it was clear that a small portion had come away, for there was now a small circular perforation, somewhat less than a shilling in size, through which the dura mater protruded, partially covered with granulations. The perforation was situated a little to the left of the middle line, and about the centre of a line drawn from the forehead to the occiput; the irregularity in the surface of the cranium prevented more accurate measurements. On the evening of the 25th inst. the nurse had just completed taking patient's temperature (102·2°), when she suddenly took an epileptiform fit, which was shortly afterwards repeated; in both these fits the head and eyes were first turned to the right, then the right arm and hand were drawn up and convulsed, and shortly afterwards the left side was similarly convulsed. A dose of chloral was given, and the convulsions were not repeated. From this date, however, patient gradually became dull and heavy, lying quiet, making no attempt to answer questions and not appearing to understand them [she had a good deal of reasoning power before this attack came on]; she also seemed distinctly deaf. On March 8th (eleven days after the convulsions) her right arm was found to be partially paralysed, exhibiting also some amount of rigidity: six days subsequently (14th inst.), the right arm had become completely flaccid and helpless, and the paralysis had spread to the right leg, which, though not absolutely devoid of movement, exhibited great defect of this. As the dura mater was now swollen and tense in the perforated area, it was incised, and about half a drachm of thick creamy pus escaped. No improvement, however, resulted in the symptoms. The cerebral substance now presented itself at the cranial opening in the form of a globular, pulsating mass.

The patient gradually became weaker, presenting ever increasing mental hebetude; she did not speak at all, and appeared hardly to understand anything that was said to her. Pupils 3 mm., equal, regular, acted well to light and to accommodation. The right arm remained perfectly paralysed and flaccid, but the paralysis of the right leg was never quite complete.

The patient gradually became more comatose, and died on March 24th, twenty-seven days after the access of the convulsions.

Autopsy (seventeen hours after death):

Cranium.—Skull-cap presented numerous irregular depressions from localised losses of substance, in both frontal and parietal regions; in parietal region, a little to the left of middle line, an opening about the size of a shilling, through which brain-tissue protruded. Dura-mater somewhat abnormally adherent to skull: a little thin recent blood clot smeared over inner surface of dura-mater at anterior part of left frontal lobe. Very little fluid in subdural space. Pia-mater over left frontal lobe somewhat hyperæmic, but both here, and over the right hemisphere, it was thin, and stripped readily. Slight opacity of arachnoid here and there in right hemisphere. Longitudinal sinus contained soft recent black clot, and lateral sinuses distinct ante-mortem, partially decolorised thrombi, which were not, however, distinctly adherent to the walls of the vessels. Inner surface of dura-mater rather firmly adherent to arachnoid over a good portion of left parietal lobe, and also, although to a less extent, over left temporo-sphenoidal lobe; on separating these membranes, thick greenish pus escaped from a rent, situated near the upper end of the left ascending parietal convolution. The portion of disorganised brain-tissue which protruded through the cranial perforation, was found to consist of the upper three-fourths of an inch of the left ascending parietal convolution, a portion of the upper part of the paracentral lobule, and a very small portion of the adjoining part of the ascending frontal gyrus. The pus which escaped was found to communicate with an abscess cavity about the size of a pigeon's egg, and containing nearly an ounce of pus, which was situated in the centrum ovale, extending towards the lateral ventricle, but not reaching this; it had hollowed out the greater part of the ascending parietal gyrus, which collapsed into it; this gyrus, however, retained its cortex (although this was superficially softened), with the exception of the upper part, which, as above mentioned, was disorganised, and protruded through the perforation. There was also a small abscess cavity in the left angular gyrus, and some superficial softening of the cortex of the left first and second temporo-sphenoidal gyri, which were smeared with pus, and a vein of some size running along the upper margin of this lobe was plugged with an ante-mortem thrombus. Ventricles not obviously dilated. Basal ganglia perfectly normal, as indeed was the right hemisphere; cerebellum, pons, and medulla also normal.

Brain (immediately after removal) = 1185 grammes.
Right hemisphere = 505 grammes.
Left hemisphere = 524 grammes (both stripped of membranes).
Cerebellum = 123. Pons = 18. Med. Obl. = 9.
Basal vessels healthy.

Thoracic and *Abdominal* viscera normal, with the exception of partial consolidation of the posterior half of the lower lobe of the left lung.

Remarks.—Though the lesions in this case were a little diffused, the case is nevertheless of interest, in that, an irritative, and subsequently destructive lesion of the left motor area was associated first with convulsions, and afterwards with paralysis of the limbs of the right side of the body. The left ascending parietal convolution was practically completely functionless, for though the cortex was not wholly destroyed, the fibres proceeding therefrom seemed quite to be so, and the lesion extended on to the median aspect, involving a portion of the paracentral lobule. The paralysis of the opposite limbs hence finds a ready explanation, whilst the convulsions to which the paralysis was consecutive, commencing on the right side, and subsequently spreading to the left, followed the course noted in electrical stimulation of the cortex. It is perhaps legitimate to associate the superficial softening of the left first and second temporo-sphenoidal gyri with the deafness from which the patient seemed to suffer; for though this symptom was difficult to disentangle from the general hebetude, it was remarked during life before deep coma supervened.

DISTRIBUTION OF ANÆSTHESIA AFTER DIVISION OF THE MEDIAN NERVE.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.

Physician to the Queen's Hospital, Birmingham.

IN the April number of "BRAIN" for last year there is a paper by Dr. James Ross upon the distribution of anæsthesia in cases of disease of the branches and roots of the Brachial Plexus. The following case, at present under my care, supports several statements in that paper. Krause's account of the distribution of the digital branches of the nerves of the hands is fully confirmed, except that in my case there was complete anæsthesia of the dorsum of the ungual phalanx of the thumb.

The anæsthesia was complete for some time after the accident, and was far more extensive than in the cases observed by Létiévant.

The patient, a medical student, was reaching a bottle of soda-water out of its case, when the bottle burst in his hand, severely cutting his right wrist in front and in the middle line. This occurred on the 25th of September last.

At the time of the accident he perceived, as it were, a great rush of blood into the hand, which felt as though it was being greatly distended, and became cold and numb.

During the dressing of the wound, and every time the pad was used to swab up the blood so as to look for a divided nerve or artery, the pain was most acute, which was probably due to the pad coming in contact with the divided nerve.

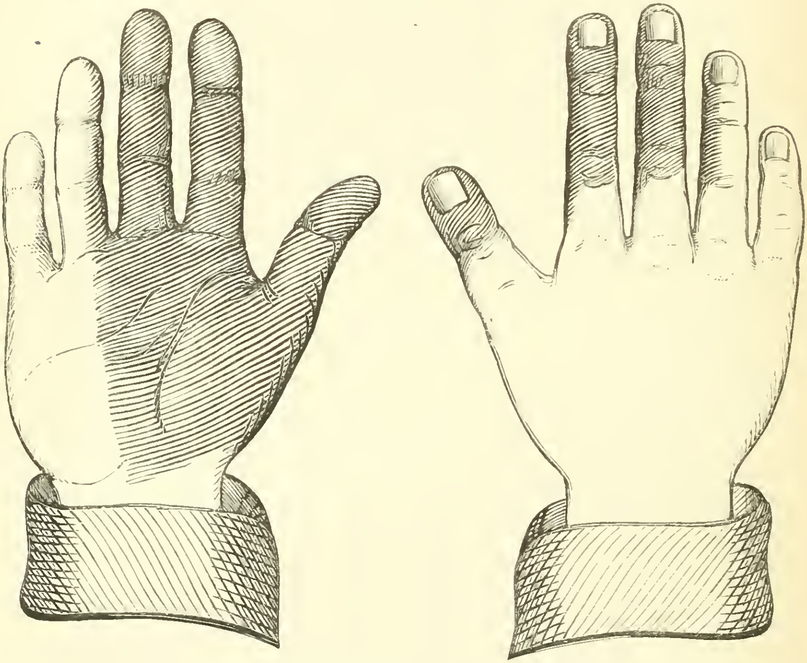
The nerve was not seen during the treatment of the wound. The hand was put in splints for three or four days; and on their removal he found that the muscles of the hand supplied by the median nerve were paralysed, and that there was total anæsthesia in the area of distribution of its sensory branches.

Three days after the accident there was total anæsthesia of the radial half of the palm and thenar eminence, the anæsthetic area being sharply defined and not gradually shading off; it extended in the palm to a line contiguous with the axis of the ring-finger.

There was also complete anæsthesia of the palmar surfaces of the thumb, index, middle and radial half of ring-finger.

On the dorsum of the hand there was total anæsthesia of the ungual phalanx of the thumb and of the second and third phalanges of the forefinger and middle finger, and of the radial halves of the same phalanges of the ring-finger.

A few days after the accident he began to feel sharp shooting-pains in the hand and fingers, which would come on suddenly and as suddenly disappear. These pains frequently prevented sleep, but after lasting for about a month they gradually ceased.



Figs. 1 and 2.—Three Days after Accident.

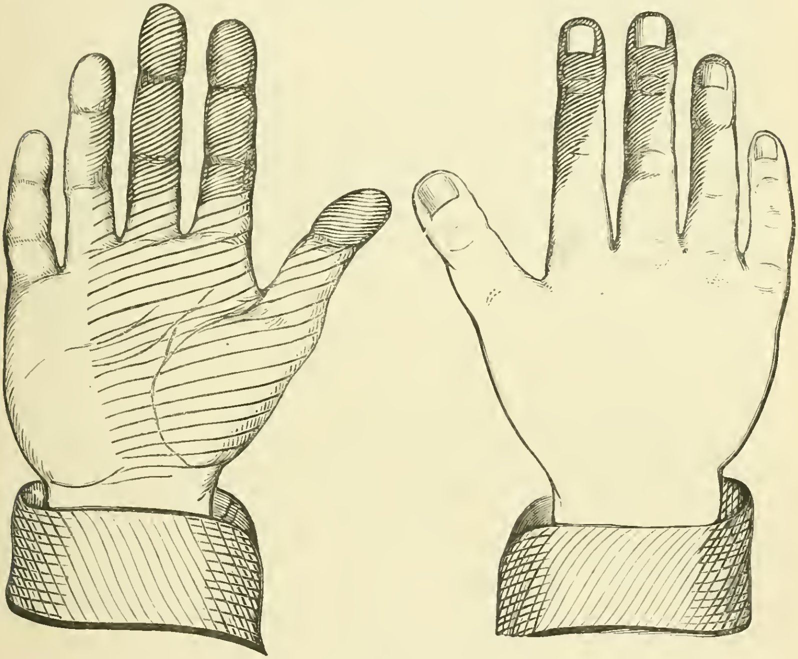
The Shaded portion represents complete Anæsthesia.

When he attempted to move the fingers he was always troubled with a sharp pain of a tearing character, which would at times extend into the forearm; this was especially acute on moving the index-finger. This pain also gradually ceased.

Since the accident there has been a great diminution of temperature in the three and a half fingers, and during the cold weather, he had great difficulty in keeping them warm. After exercise he frequently noticed the little and half the ring-finger and the inner part of the hand to be covered with perspiration, while the rest of the palmar surface of the hand was quite dry.

At the present time (February), nearly five months after the accident, there is a scar in front of the right wrist passing obliquely across the tendon of the flexor, carpi radialis and median nerve. The scar is very sensitive when touched. A small bulla, containing blood, is seen on the inner side of the terminal phalanx of the index-finger.

No changes in the nails are to be observed. He has recovered power to a considerable extent, being able to write with the right hand; but the grasp is much weaker than on the left side.



Figs. 3 and 4.—Five Months after Accident.

The portion thickly Shaded represents the Anæsthesia; the thinly Shaded portion Hyperæsthesia.

There is wasting of the thenar eminence on the right side. The circumference of the base of the right thumb being five inches, that of the left, five and a half. The response of the muscles of the hand supplied by the median is much diminished to faradism. To galvanism the response is increased, the contractions being slow and prolonged, the anodic closure contraction predominating.

The distribution of the anæsthesia at the present time, nearly five months after the accident, is as follows. The area of the right palm previously anæsthetic is now everywhere hyperæsthetic, a slight touch causing pain, the pain radiating into the fingers.

The hyperæsthesia extends over the palmar surface of the first phalanx of the thumb and encroaches on the first phalanges of the forefinger, middle finger, and ring finger.

The skin of the rest of the phalanges are still anæsthetic on slight pressure, but hyperæsthetic on deep pressure, the pain radiating into the fingers.

On the dorsum of the hand the following changes have occurred :

The anæsthesia previously present on the dorsal aspect of the ungual phalanx of the thumb has disappeared. The only alteration in the other fingers is that the anæsthesia of the middle phalanges of the fore and middle fingers extends now only over the radial half of the phalanges, the ulnar half being normal.

A CASE ILLUSTRATING THE CONDITION OF THE NERVOUS SYSTEM AFTER AMPUTATION OF AN EXTREMITY.

BY WILLIAM DUDLEY, M.B. (LOND.),

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The published cases in which have been described lesions of the nervous system as the results of amputation, are so few, that the following seems worthy of record.

Adolph K——, 39, married, was admitted into the West Riding Asylum, Feb. 28th, 1885.

History (obtained on admission).—Patient, who is a Pole, according to his own account, lost his left leg, through bullet-wounds received in 1864. During the past three years he is said to have been excitable, and to have behaved frequently in a brutal manner towards his wife and his employés. About five weeks ago he was thrown from a trap, and sustained a fracture of the right leg. Symptoms of insanity were first observed while he was confined to bed with the broken leg.

On admission, there was an ununited fracture of the right leg. The left leg had been amputated at the junction of the upper and middle third of the thigh.

The case was an ordinary one of general paralysis, and death from exhaustion occurred on March 25th, the fatal termination being hastened by suppuration at the seat of fracture, and by large bed-sores.

At the autopsy, 26 hours after death, the fractured ends were found necrosed; the surrounding tissues were infiltrated with pus; there was fibroid induration of the lungs, and deep congestion of the lower lobes; there was slight atheroma of the aorta; the kidneys were slightly cirrhosed.

The brain showed the appearances ordinarily met with in a moderately marked advanced stage of general paralysis, viz. atrophy, excess of serum (five ounces being collected), opaque, thickened and tough membranes, with cortical adhesions of small superficial extent and slight depth.

The ascending parietal convolution and the postero-parietal lobule of the right side were smaller than those of the left. The right ascending frontal was crossed at 1½ in. from the longitudinal fissure by a deep sulcus. Its knee was much smaller than that of the opposite side. A naked-eye examination revealed nothing abnormal in the basal ganglia, the cerebellum, the pons and medulla.

In sections taken from the ascending frontal, the ascending parietal, and the postero-parietal lobule of the right side the cortex is seen to be of average depth, and the different layers of cells to be distinct. The nerve-cells do not appear to be diminished either in size or in number; some of them show pigmentary degeneration, and the ground substance is more deeply stained than occurs in the normal condition, these changes being similar to those usually met with in general paralysis, and may probably have no relation to the loss of the left lower extremity.

Spinal Cord.—In the cervical and dorsal regions there is no appreciable difference in the grey or white matter of opposite sides. The cells are equally numerous, and have undergone degeneration to a similar extent. The white matter is healthy.

In the dorso-lumbar region no decided difference in the two sides can be detected. At the thickest part of the lumbar enlargement a difference in size of the anterior cornua is observable, the right being the larger. Microscopically, the cells of the right side are found to be a little more numerous than those of the left.

About one inch from the lower extremity of the cord, a difference in the sectional area of the grey matter of the two sides is obvious, the transverse measurement of the right anterior cornu being to that of the left in the proportion of 3 to 2. From this level downwards the difference diminishes, but can still be seen even when the diameter of the cord does not exceed a line. The right posterior cornu is very slightly larger than the left.

At the level at which the difference in size of the anterior cornua is most marked, a still more obvious difference in the number and size of the cells is seen upon microscopic examination, about 15 to 18 fairly plump cells being present in a transverse section on the left side, whereas there are about 40 to 45 on the right; moreover, the majority of those on the left side are distinctly smaller than those on the right, so that if the 30 largest cells in both cornua be taken, about 24 or 25 will belong to the right side. The antero-lateral and postero-lateral groups are those in which the difference is most evident. No morbid change is detected in the white matter. Near the lower extremity of the cord it is ragged and ill-stained, probably owing to its having been sodden with serum.

Throughout the cord, side by side with cells which appear perfectly healthy, are numerous cells which have undergone pigmentary degeneration. Some of these appear to be of normal size, and show a distinct cell-wall with numerous well-stained processes; but in place of healthy protoplasm, which readily takes up most staining reagents, the greater part of the interior of the cell is occupied by yellow granular pigment, which does not stain at all, or but very faintly. Other cells have a rounded outline, and are frequently pear-shaped, and appear swollen, from the presence of a mass of pigment, while the very small amount of healthy protoplasm which remains appears as if pushed away to the narrow end of the cell, from which a well-stained process is usually seen to extend, the other processes being faintly stained, or invisible. Other cells show a cell-wall enclosing nothing but coarsely granular pigment, and have only few and feebly-stained processes. These cells are generally, but not always, smaller than the more healthy cells in their immediate neighbourhood. Finally, there are found masses of pigment, with neither protoplasm nor processes, and these are in all probability the remains of cells which have entirely lost their function. These extremely degenerate elements are much more numerous in the lumbar region, and on the left side; but judging from their number in this situation, it is probable that here some cells have disappeared entirely, so as to leave no trace behind.

Within the cord, the fibres of the anterior nerve-roots seem to be more numerous on the right than on the left side, at the level at which atrophy is well-marked. Among the fibres of the left side are here and there to be seen axis cylinders, represented by a line of dots or by a row of short irregular broken lines, and deeply-stained granular masses are also found in this situation, which probably represent degenerate fibres.

Transverse section of the left anterior crural nerve show bundles of very fine nerve-fibres scattered irregularly between healthy fibres. These fine fibres are seldom isolated. They occur in groups containing from two or three up to thirty or forty, and the groups appear, on section, of the most varied shape. Some of the fasciculi contain more of the fine fibres than others do; none appear to be quite free. The fine fibres stain much more deeply than the healthy ones. Their diminished size appears to be due mainly to a much-attenuated medullary sheath. Their diameter varies considerably, but the majority are about one-third or one-fourth that of the healthy fibres. A large amount of adipose tissue exists between the fasciculi. It is to be regretted that the left anterior crural nerve was the only one preserved.

A CASE OF ACUTE MANIA, IN WHICH THERE WAS COMPLETE REMISSION OF THE SYMPTOMS DURING A TEMPORARY PLUGGING OF THE SINUSES.

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M. S., æt. 41, married, housewife, admitted into the West Riding Asylum, July 1882, suffering from an attack of acute mania.

The attack began a week previous to admission, to account for which there was no special illness, shock or trouble.

About sixteen months before, she had been confined, and had suckled her child up to the date of the present illness. She was of good moral character, and a steady hard-working woman.

She is said to have suffered from depression and nervousness for some weeks before admission. She has had ten children, seven of whom are alive and well. After her last confinement, catamenia were regular.

Family History.—A maternal uncle died in this asylum, and her grandfather died insane. No history of epilepsy, apoplexy, or phthisis.

On admission, the patient was in a state of acute maniacal excitement; she struggled incessantly, and was quite incoherent and hoarse from continuous raving. She was the subject of delusions, stating that there was war in the village where she lived, and wildly shouted, "John the Baptist is my son." She appeared alarmed at times, and would cry out that the children in the house were being killed.

Her physical condition was fairly good, pupils equal. The right mamma was larger than the left.

For some weeks after admission the patient continued in the same state of restlessness and maniacal excitement, causing her to become pale, anæmic, and much reduced.

August 11th.—There was noticed an appearance of redness and fulness of the left ear, which was found to be hot, and suggestive of commencing hæmatoma auris.

September 12th.—The left ear commenced to shrink, but hæmatoma of the right ear had developed. The patient was more excited than ever, continually abusing those around her, wrestling with the attendants, and violent to every one that approached her.

January 15th.—A marked change came over the patient. She became suddenly quiet and composed, talking in a rational manner, and showing no sign of mental weakness. She was slightly feverish, however, with a throbbing pulse, and there was noticed in the region of the parotid gland a distinct swelling and tenderness.

16*th.*—The parotid swelling was found to have subsided, but over the mastoid cells on the right side a painless tumefaction had appeared. There was also in the right orbital region a general puffy and œdematous condition. She complained of occipital headache.

18*th.*—The swelling in the orbital region was considerably diminished, and the patient still continued quiet and rational. She complained, however, of pain in her throat, which was found to be slightly congested.

February 2nd.—The œdematous condition was found to have quite subsided, and the tumefaction over the mastoid cells had completely disappeared, whilst the patient herself had just as suddenly relapsed into her old ways of disorderly excitement. She was garrulous and abusive as formerly, and in much the same state of persistent excitement she has continued up to the present time, taking her food badly and sleeping little, whilst her physical condition has become much reduced.

It is of interest to note in this case, that the sudden remission of the maniacal symptoms coincided in point of time with the appearance of the œdema of the orbital region, and over the mastoid cells; whilst the disappearance of this œdema was attended with just as sudden a relapse into the maniacal state.

Critical Digests.

RECENT OBSERVATIONS ON DEGENERATION, AND ON NERVE TRACTS IN THE SPINAL CORD. A CRITICAL ACCOUNT.

BY J. N. LANGLEY, M.A., F.R.S.

Fellow and Lecturer of Trinity College, Cambridge.

1. *Langley and Sherrington.* "Secondary Degeneration of Nerve Tracts following removal of the Cortex of the Cerebrum of the Dog." 'Journal of Physiology,' v. p. 49. 1884.
2. *Pitres.* "Recherches anatomo-cliniques sur les scléroses bilatérales de la moelle épinière consécutives à des lésions unilatérales du cerveau." 'Arch. de Physiologie normale et pathologique,' p. 142. 1884.
3. *Löwenthal.* "Des Dégénérations secondaires de la moelle épinière consécutives aux lésions expérimentales médullaires et corticales." 'Dissertation.' Genève (Schuchardt), 1885.
4. *Sherrington.* "On Secondary and Tertiary Degenerations in the Spinal Cord of the Dog." 'Journal of Physiology,' vi., p. 176. 1885.
5. *Homén.* "Experimenteller Beitrag zur Pathologie und pathologischen Anatomie des Rückenmarkes." 'Fortschritte der Medicin' (No. 9, May), p. 267. 1885.

The broad outlines of our knowledge of the nerve tracts of the spinal cord, we owe very largely to the observations on degeneration, made by Turck[1] in 1851 to 1853, and by Bouchard[2] in 1866. Since 1866 there have been many observations confirming in the main the results of Bouchard, but new results have come slowly and sparsely. The principal additional facts are perhaps those noted by Schiefferdecker[3] in 1865, and by Singer[4] in 1881. The general acceptance of these results is due chiefly to the work of Flechsig[5] in 1876, on the times at which the various nerve-fibres in the spinal cord acquire their medulla. The degeneration method and Flechsig's method gave similar results, and the mass of detailed information brought forward by Flechsig produced a belief not accorded to the previous observations on degeneration. Moreover, Flechsig's description was much more complete than that of his predecessors.

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[1] Türck, 'Sitzungsb. d. Wiener Akad.,' Bd. vi. p. 288, 1851; Bd. xi. p. 93, 1853; also in Bd. xiv. p. 329, 1855.

[2] Bouchard, 'Archives Générales de Médecine,' vi. ser. t. vii. pp. 272, 441, 561; t. viii. p. 273 (1866).

[3] Schiefferdecker, 'Virchow's Archiv,' Bd. 67, p. 542, (1876).

[4] Singer, 'Sitzungsb. d. Wiener Akad.' Bd. 83, p. 390, (1881).

[5] Flechsig, 'Die Leitungsbahnen im Gehirn u. Rückenmark des Menschen,' (Leipzig) 1876.

But there still remain many points undetermined. I propose in the following paper to discuss these points on the basis of the observations made in 1884 and in 1885. In doing so it will be necessary, for the sake of clearness, to refer to many of the earlier observations.

We will first take the changes which can be observed in secondary degeneration, since some of the discrepancies between the accounts of different writers are due to the different methods employed, and to the different times after the lesion at which the cord is examined.

The characters of secondary degeneration are the same, whether it is caused by lesion of the spinal cord or of the brain. We will, then, refer to cases of secondary degeneration produced by either cause. And for convenience we will take separately the changes observed according as the spinal cord is, (1) cut across in the fresh state and examined straightway; or (2) hardened in potassium bichromate or other similar agent and then cut across and examined; or (3) hardened, and sections stained and examined with the microscope.

1. *Spinal Cord examined in the fresh state.*—In a transverse section of the fresh cord, the degenerated area has a more milky-white appearance than the rest of the white substance; this is apparently due to a change in the medulla of the nerve fibres. According to Löwenthal, this change can be seen as the result of section of the cord, or of injury to the motor area of the brain, at all times between three and twelve weeks after the lesion. His observations on the result of section of the cord do not extend beyond these limits. He has made one observation at an earlier date on the effect of cortical brain lesion. The cord was examined fifteen days after the injury. A milky-white spot was present in the lateral pyramidal tract in the cervical region; whether it was present or not in the rest of the cord he does not mention.

Sherrington was unable to observe in the dog the "milky" degenerative change in the lateral pyramidal tract, after injury to the motor area of the cortex, earlier than the fifth week. In three to four months, according to Sherrington, the spot becomes less white, and then greyish, at the same time acquiring a more transparent and gelatinous look. This is the state in which it has most commonly been described in the fresh cord.[1] Finally, it becomes unrecognisable in the fresh cord, although after the cord is hardened it may still readily be seen.

2. *Spinal Cord hardened in Potassium Bichromate.*—The degenerated area in this case appears as a yellowish spot of lighter tint than

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[1] For a *résumé* of the descriptions which have been given of the appearance of the area of degeneration in the fresh cord, cf. Löwenthal, op. cit. (3), p. 84.

that of the rest of the cord. Singer[1] observed this in the cerebellar tracts, and in the columns of Goll of the dog, throughout the cord, twelve days after section in the lower dorsal region.

Löwenthal describes the descending degeneration as being thus visible, though not very distinctly, twenty-one days after hemisection of the cord of the dog in the cervical region, and as being much more obvious three months after the injury.

Sherrington finds that after destruction of the motor area of the cortex, the yellowish spot of degeneration is visible in nine days from the 1st cervical to the 2nd lumbar nerve-roots; but is, up to a limit of three to six months, the more distinct the longer the degenerative changes have been going on. Towards the end of this time it becomes smaller. In degenerations of longer date than six months the change is less distinct. It is not quite certain whether long-standing secondary degenerations ever become quite unrecognisable in the hardened cord on macroscopic investigation; they are, however, in their later stages more easily recognised under the microscope.

3. *Examination of sections of the Spinal Cord under the Microscope.*—Homén[2] in 1883 found in the dog that, after hemisection of the cord in the lower dorsal region, degenerating fibres were present in the cord *seven* days after the injury both above and below the place of section. In these fibres he found, as a rule, the neural axis (axis cylinder) only affected. In eleven days the number of fibres with altered medulla was greatly increased. In twenty-one days the neuroglia cells were more conspicuous, and some of the nerve-fibres had disappeared. In twenty-nine days the general connective tissue-substance between the nerve-fibres showed signs of alteration.

Homén in his more recent paper (5) again considers the time after hemisection of the cord at which degeneration can first be made out, and the nature of the degenerative changes. He finds that the degenerative changes do not begin at the same time in the various nerve tracts. In *three* days altered fibres can be seen in the posterior columns,[3] and can just be made out in the lateral pyramidal tract; in five days the degeneration in the lateral pyramidal tract is distinct; in seven days altered fibres are present in the cerebellar tract. During the first few days of the degenerative change, the number of affected fibres rapidly increases; how long this increase goes on is not stated.

For detecting the early stages of degeneration Homén recommends staining with acid-fuchsin. As we know from Schiefferdecker and others, the first change seen in the nerve-fibres is that the neural axis becomes somewhat swollen and granular, and stains less than normal with carmine. Homén finds that acid-fuchsin stains the degenerating neural axis much more deeply than the normal, and of a dark-red colour; so that the degenerating fibres are much more obvious than in specimens stained with carmine.

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[1] Singer, op. cit.

[2] Homén, 'Comptes Rendus,' No. 23, 1883. Schiefferdecker (op. cit.) did not find any altered fibres seven days after section of the cord of the dog in the lower dorsal region; fourteen days after section altered fibres were present, the medulla of the nerves stained slightly, the neural axis stained less than normal. Sherrington describes altered nerve-fibres in the cord nine days after cortical lesion in the dog.

[3] He finds that the change is distinct through the whole of posterior column above the place of section.

As the myelin of the nerve-fibres becomes affected, it stains slightly with carmine, and becomes with difficulty or not at all distinguishable from the neural axis. When acid-fuchsin, or hæmatoxylin after Weigert's method, is used, the inner part of the myelin first blends with the neural axis, the outer part staining as before—bright red with acid-fuchsin, black with hæmatoxylin—later, in three to eight weeks in different fibres, the outer part of the myelin sheath also blends with the axis, forming a mass which is nearly homogeneous, and stains but little. As this is going on, the fibres split up, and the separated swollen portions form colloid bodies, which are gradually absorbed.

Thus it appears that the neural axis is first affected, then the medulla, and lastly the connective tissue. The hypertrophy of the connective tissue is probably due to the irritative action of the substance of the dead and disintegrating nerves.

That the connective tissue is only secondarily affected has been laid stress on by nearly all observers.[1] When a few fibres only degenerate, the connective tissue may be unaffected: in such cases, degeneration can only be seen during the process of breaking down of the nerve-fibres; if the fibres are scattered, the degeneration can only be seen under the microscope.

In the early stages of connective tissue change, Deiters' cells are fairly numerous and conspicuous, and there are many granular cells and leucocytes; according to Bouchard, the blood-vessels are atheromatous. In the later stages these characters largely disappear; the connective tissue remains in abnormal amount, and is usually said to consist of a network of fine fibres. Sherrington and myself found it to consist almost wholly of fine semi-elastic fibres, not anastomosing with one another, and staining with carmine. We considered, too, that the connective-tissue "scar" diminished in time in staining power, and hence became more difficult to recognise.

The following may be taken as a rough summary of the chief points connected with the observation of secondary degeneration, although no doubt several of the statements are open to discussion. Secondary degeneration is best observed with the microscope four to five weeks after the injury. The degeneration can be made out near the lesion in a week or less. It spreads fairly rapidly—probably in less than twenty-four hours—along the whole course of the nerve-fibres. It can be earliest seen by the alteration in the neural axis, a day or two later by the change in the myelin sheath also. In one to two weeks it can be observed by the eye in the cord hardened in potassium bichromate. In about three weeks[1] it can be seen by the eye in the fresh cord. The neuroglia now begins to increase, and the nerve-fibres to be absorbed, although probably none disappear for some weeks. In consequence of these changes, the degeneration is, from the sixth week onwards, more and more noted under the microscope by the change in the neuroglia, and less and less by the presence of altered nerve-fibres. In the lateral pyramidal tract altered nerve-fibres are seen for three to four months. The length of time after the lesion during which the area of degeneration can be detected by the "scar," varies with the number of fibres which have degenerated; in the lateral pyramidal tract the scar can be seen for a year, and probably for much longer. The degeneration can be seen in the fresh cord for four to five months; during the last six weeks or so of this time, it changes from milky-white to greyish. Possibly in the cerebellar tract the change takes place earlier. In time (? six months) no change can be seen in the fresh cord. In the cord hardened in potassium bichromate, the area of degeneration becomes more distinct for four to six months; later than this it becomes less and less distinct; in the lateral pyramidal tract it may be seen for about a year. In its earlier stage, the area of degeneration may appear larger than it really is, in consequence of a spreading out of the connective tissue change; in its later it becomes smaller, in consequence of a retraction (? partial disappearance) of the connective tissue.

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[1] *e.g.* Schiefferdecker, op. cit. Kahler and Pick, 'Arch. f. Psychiatrie,' Bd. x. pp. 179 and 279 (1880); Schultze, 'Arch. f. Psychiatrie,' Bd. xiv. p. 358 (1883).

Whilst all observers have paid considerable attention to the time after the lesion at which secondary degeneration makes its appearance, comparatively little attention has been paid to the time at which the degeneration of the nerve fibres is complete. But this is a point of some importance. According to Löwenthal, the degenerating areas in the region of the lateral pyramidal tract, which result from sections of the cord in the cervical region, consist chiefly of altered fibres three months after the section. He finds, too, that the number of altered fibres is greater in twelve weeks than in six, and greater in six weeks than in three. Sherrington describes a few altered fibres as being present in the lateral pyramidal tract seven months after cortical lesion. But from the descriptions of most observers, we should gather that some fibres disappear altogether in about a month. Homén, in three weeks, finds spaces from which fibres have disappeared; but it is not clear whether the fibres had been absorbed from these spaces, or simply dropped out of the section. If fibres do disappear so early, and if Löwenthal's statement regarding the increase in the number of altered fibres for two to three months after the lesion is correct, it follows that some fibres have disappeared, whilst others have scarcely begun to degenerate. This seems extremely unlikely, if all the fibres are undergoing true secondary degeneration.

It seemed possible that there might be some differences between descending and ascending degeneration in these respects; but I cannot find that any one who deals with both degenerations points out any difference. The only clear statement as to the time of disappearance of nerve-fibres in ascending degeneration which I have been able to find is given by Singer. According to him, in *young* dogs after section of the cord in the dorsal region, the nerve-fibres of the cerebellar tract and of the medial part of the posterior column completely disappear in four to five weeks. In the adult dog, the process is slower; unfortunately he does not say how much slower. There seems to me, however, to be *primâ facie* ground for thinking that, at any rate in the pyramidal tract, some fibres do not begin to degenerate until several weeks after section of the cord.

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[1] In man it is often difficult to observe any change in the fresh cord; possibly this may be on account of the time which elapses before the cord is examined.

Turning now to the nerve-tracts in the cord, we may first consider—

The Pyramidal Tracts.—Flechsig mentions that in the lateral pyramidal tracts there are a certain number of fibres which do not come from the opposite pyramid. But in addition to these fibres, it is probable that the lateral pyramidal tract, as described by Flechsig, contains many other fibres which do not belong to this tract.

Bouchard in 1866 noticed that in man, the descending degeneration in the lateral column was considerably greater after injury to the medulla than after injury higher up in the brain.

Bouchard[1] describes the area of degeneration which appears in the cord in the posterior part of the lateral column after injury to the medulla, as extending to the surface. Since the cerebellar tract occupies the periphery of this part of the cord, and does not undergo descending degeneration, Bouchard's statement regarding the peripheral strip probably rests on a misinterpretation of the appearance. According to Schiefferdecker, the sclerosis may extend beyond the area of the degenerated fibres; to this the sclerosis seen by Bouchard at the periphery of the cord was probably due.

The fibres which degenerated after injury to the medulla, but not after injury to the brain, were situated on the lateral border of the pyramidal tract. These fibres he called "fibres commissurales antérieures longues." This observation has passed with little or no notice.

A somewhat similar difference, however, is described by Löwenthal, as occurring in the dog, after section of the cord in the cervical region and after removal of the motor area of the cortex. According to Löwenthal, the long commissural fibres lie between the cerebellar tract and the lateral pyramidal tract; they are chiefly large fibres; whereas, as we know, the pyramidal tract fibres, like those of the pyramid, are chiefly moderate-sized and small. Löwenthal points out also that, after section of the cord in the cervical region, the degenerated area in the lateral column has a fairly sharp boundary on its lateral edge. In degeneration after removal of the cortex of the brain, this is certainly not the case, the degeneration becomes quite diffuse as it approaches the cerebellar tract.

Sherrington also has come to the conclusion that, at any rate in the cervical region of the cord, the real pyramidal tract occupies a part only of the pyramidal tract as figured by Flechsig, This conclusion is, however, based upon a comparison of the area of degeneration which he obtains in the dog, with the area of non-medullated fibres seen in the developing cord of the ape, and with the area of the pyramidal tract as given by Flechsig in the cord of man. The basis is somewhat uncertain. Proceeding, however, in this way, he concludes that the ventral and the latero-dorsal parts of Flechsig's pyramidal tract do not degenerate from cortical lesions, and hence are not continuations of the pyramid fibres.

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[1] Bouchard, op. cit. t. vi. p. 570.

On the whole, it seems probable that the pyramidal tract is much smaller than that given by Flechsig, that it does not form a compact bundle of fibres, but on its anterior (ventral), lateral and posterior (dorsal) borders, it becomes diffuse, its fibres being mixed with other fibres, many of which, especially on its lateral border, degenerate on section of the spinal cord. In its inner (mesial) side its fibres, as described by Flechsig, become mixed with the small fibres of the "boundary layer." It will be noticed that, as far as Löwenthal's experiments extend, the "anterior long commissural fibres" might be "commissural"[1] fibres of the cord. From Bouchard, however, we should conclude that they run from the medulla to the various segments of the cord, and hence we might call it the descending medullary tract.

Bilateral Degeneration of Lateral Pyramidal Tract in consequence of Unilateral Cortical Injury.—It has been known for some time, that in man an injury confined to one side of the cord, might cause a degeneration in the lateral pyramidal tract of the opposite as well as in that of the same side of the cord. Charcot[2] suggested as an explanation of this, that part of the lateral pyramidal tract crossed in the anterior commissure, especially in the upper dorsal region, to the lateral tract of the opposite side. Thus the fibres would first cross in the pyramidal decussation, and then again in the anterior commissure.

A case of bilateral degeneration in the cord of the dog, as the result of unilateral cortical injury, was described in 1880 by François-Franck and Pitres.[3]

Moeli[4] in 1883 found after unilateral injury to the mid-brain in dogs, that besides the degeneration of the pyramidal tract of the opposite side there was a degeneration of a few scattered nerve-fibres in the pyramidal tract of the same side.

An instance in the ape has also been described by Schäfer[5] (1883); the examination was made seven months after the cortical injury. In the cervical region of the cord there was sclerosis in both lateral pyramidal tracts, the area of the two sides being about equal; but the sclerosis was less in intensity on the same side as the lesion, and on this side it could not be traced beyond the cervical region.

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[1] Cf. p. 106.

[2] Charcot, 'Leçons sur les localisations dans les maladies du cerveau et de la moelle, recueillies par MM. Bourneville et Brissaud,' p. 252 (Paris, 1876–1880).

[3] Francois-Franck et Pitres, 'Progrès Méd.' p. 146 (1880).

[4] Moeli, 'Arch. f. Psychiatrie,' xiv. p. 173 (1883).

[5] Schäfer, 'Jour. of Physiology,' vol. iv. p. 324 (1883).

The bilateral degeneration of the pyramidal tract has received a good deal of attention from recent observers. In no case apparently does unilateral brain-injury cause degeneration of the opposite pyramid,[1] so that whilst one pyramid only degenerates, there is degeneration in the region of both the lateral pyramidal tracts in the cord. We will, for lack of a better term, call the degeneration which occurs on the side of the cord opposite to the sound pyramid the *re-crossed degeneration*.[2]

Pitres examined the spinal cord in forty cases of unilateral brain-injury in man. In ten of these there was on both sides sclerosis of the lateral pyramidal tracts. In some cases this could only be made out on microscopical examination. In six out of the ten cases the sclerosis was symmetrical and equal on the two sides, and on both sides it occupied a larger area, chiefly in the ventral direction, than that occupied when sclerosis occurred on one side only. In the remaining four cases the re-crossed degeneration was of less intensity than that of the opposite side. According to Pitres, the re-crossed degeneration stretches throughout the cord, reaching the surface of the cord in the lumbar region. No relation could be made out between the presence or absence of a re-crossed degeneration, and the presence or absence of a degeneration in the medial part of the anterior column (the direct pyramidal tract, Türck's column). In six cases the direct pyramidal tracts showed some sclerosis; this, whilst varying in amount, was in all cases slight, and in one case only could it be traced in the lower dorsal and in the lumbar region. In three cases there was sclerosis in *both* direct pyramidal tracts.[3]

Pitres considers the re-crossed degeneration to be due to the degeneration of fibres coming from the pyramid of the same side; *i.e.* he extends to these fibres the view advanced by Bouchard and by Flechsig with regard to the direct pyramidal tract.

There are on this view four ways by which the fibres of each pyramid can proceed to their ending in the cord. They proceed along (1) the lateral pyramidal tracts of the opposite side; (2) the lateral pyramidal tract of the same side; (3) the direct pyramidal tract of the same side; (4) the direct pyramidal tract of the opposite side. Most commonly all the fibres take the first course; not infrequently they run both in the first and second courses; less frequently some fibres proceed by the third, the greater number running either in the first or in the first and second course; rarely some fibres may in addition take the fourth course. If this description is the true one, the term "re-crossed" degeneration, which I have used above, is obviously inapplicable; but there are reasons, which will be mentioned later, against accepting Pitres' views. Charcot's suggestion, that the fibres of one lateral tract cross to the other by the anterior commissure has, as Pitres points out, no satisfactory basis, for no degenerated fibres have as yet been described as occurring in the anterior commissure.

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[1] According to Sherrington a *few* scattered degenerating fibres may be present in the opposite pyramid. But I gather from his description that the number of these is wholly insufficient to account for the degeneration in the cord.

[2] This implies that the degeneration is the consequence of a previous degeneration on the opposite side of the cord—a view which though probably is not proved; the term "re-crossed pyramidal tract" used by Sherrington, is, however, still less satisfactory. A neutral term would have been best, but I could think of none.

[3] In all of these cases the degeneration had advanced to the stage of sclerosis.

Bilateral degeneration in dogs after unilateral cortical injury has been described both by Löwenthal and by Sherrington.

Löwenthal mentions it as occurring in two out of about twenty cases.[1] In each of the two the degeneration (sclerosis) was diffuse, and could not be traced farther than the mid-dorsal region. Sherrington found bilateral degeneration in each of twelve cases observed three to twelve months after the lesion, but found unilateral degeneration only, in two cases observed seven and fourteen days respectively after the lesion. The re-crossed degeneration appeared to him to be always somewhat less advanced than the degeneration on the opposite side of the cord. Hence he concludes that the re-crossed degeneration begins three or more weeks after the degeneration in the opposite lateral pyramidal tract. The conclusion is somewhat hasty; for in the two cases in which re-crossed degeneration was absent, it might not have occurred had the animal been kept alive; and further, Sherrington does not appear to be quite certain that the nerve-fibres in the recrossed degeneration were really in an earlier stage of degeneration than those on the opposite side of the cord. It may be noted, however, that if his conclusion is right, it disposes at once of the views both of Charcot and of Pitres as to the origin of the fibres in the re-crossed degeneration; for if the fibres which are affected in the re-crossed degeneration were directly continuous with the pyramid fibres, they would certainly not degenerate weeks later than the rest of the pyramid fibres in the lateral pyramidal tract.

Sherrington also finds that the re-crossed degeneration does not usually diminish regularly in its course down the cord, but is greater in the upper lumbar and lower dorsal region than in the mid-dorsal region, and is commonly greater between the third to seventh cervical nerve-roots—where, indeed, it is earliest seen—than between the first to third cervical nerve-roots. If this is the case, the bilateral degeneration cannot be due to a crossed and an uncrossed portion of the pyramid, and the re-crossed degeneration cannot be due to pyramid fibres. It is on this account that I have used the term "re-crossed degeneration."

If the pyramids send no fibres to the lateral column of their own side, some other explanation of bilateral degeneration must be sought for. Let us consider in what other way it could be brought about. Since in the dog there is no evidence of a direct pyramidal tract, we have for the dog the simple scheme, that all the pyramid fibres running to the cord decussate in the pyramidal decussation, and run down the cord in the lateral pyramidal tract of the opposite side. Since these fibres end in the grey substance of the cord, it is quite possible that their degeneration should cause more or less pronounced degeneration in some parts of the grey substance. And in fact one or two cases have been described of atrophy of cells in the anterior cornu in consequence of degeneration of the lateral pyramidal tract, and of atrophy of Clarke's column in consequence of degeneration of the posterior roots. And there is some evidence that in man the grey substance in lateral sclerosis is not infrequently affected in a less degree; for on the non-hemiplegic side it is not uncommon to find increase of reflexes, or general weakness without any discernible change in the columns of that side of the cord. These cases, although admitting of other explanation (see below), are most satisfactorily explained by supposing that the grey substance of the cord has been affected by the degeneration of the pyramid fibres. If the change proceeds further than this, viz. to degeneration, it is to be expected that some fibres proceeding from the grey substance to the columns of the cord, *i.e.* connecting fibres, will degenerate. Such fibres, we may fairly conclude—bearing in mind the assumption with which we started—are the fibres of the re-crossed degeneration.

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[1] It may be noticed that Homén (1883), in his account of the effects of hemisection of the cord in the dog, makes no mention of bilateral degeneration.

But if the disappearance of the pyramid fibres on one side can affect the grey substance sufficiently to lead to the degeneration of connecting fibres on the opposite side, we should certainly expect that it would cause some degeneration of connecting fibres on its own side. There seems to me to be some evidence that this does take place. We have seen above (p. 97), that there is some reason to believe that within the area of the lateral pyramidal tract some fibres show signs of degeneration very much earlier than others. This is not easy to explain, if all the fibres are the direct continuation of the fibres of the pyramid; whereas if the fibres which are first affected are pyramid fibres, and the later ones connecting fibres of the cord, the protracted period of degeneration presents no difficulty.

Sherrington and myself have used the term "tertiary" (cf. p. 107) to denote that degeneration which is brought about by a change in grey substance as a consequence of nerve-fibres in it degenerating. We might then state the conclusions of the foregoing argument in the following way: injury to one lateral column of the cord (or unilateral injury to the motor area of the brain) causes secondary degeneration in the crossed pyramidal tract, and not infrequently tertiary degeneration of connecting fibres of the cord within the area of both crossed pyramidal tracts.

This view affords a basis for understanding how the re-crossed degeneration can, as described by Sherrington, be greater in the lower dorsal than in the mid-dorsal region; why it occurs later than the 'normal' degeneration; why on both sides of the cord fibres in an early state of degeneration are found long after the injury; and why in bilateral degeneration the area is often, as described by Pitres, greater than in unilateral degeneration. It does not satisfactorily explain how the sclerosis can be—as described by Pitres in certain cases—equal in intensity on the two sides of the cord.

But with all this it must not be forgotten that the statements on which the view rests require confirmation; and further, that so far no relation has been observed between bilateral degeneration and atrophy of grey substance. I have stated the view with some fulness, on account of its importance, if true; but I need hardly say that I regard the questions here raised as at present quite open.

One other question of importance arises out of bilateral degeneration. This is whether each pyramid is connected with the grey substance of both sides of the spinal cord.

Flechsig, in his "Plan des menschlichen Gehirns,' figures the "direct" pyramidal tract as ending in the grey substance of the same side of the cord; those who accept Pitres' view of the meaning of bilateral degeneration might consider that here, too, each set of fibres ends in the grey substance of its own side.

That is to say, the extent of the uncrossed tracts of the pyramid would be a measure of the number of its fibres which end in the cord on the same side as it. The variability of the course of the pyramid fibres in the cord would be associated with a variability in their termination.

It is clear that there is no necessity that this should be the case. All the fibres of one pyramid might run in one lateral column, and yet end in the grey substance of both sides of the cord; and on the other hand, the fibres of one pyramid might run in both lateral and in both anterior columns, and yet end in the grey substance of one side only of the cord.

And that this is the case seems to me to be in the highest degree improbable. It requires a great deal of evidence even to make it likely that the individuals of one species vary so much, that in one the cortex of the brain is connected with one side only of the cord, and in another is connected with both sides; that, in fact, in different individuals the end stations of homologous fibres are indifferently on the right or on the left side of the body.

What, then, is the proof of this variation? It is partly that the pyramid fibres are said to vary in their course in the cord; this, even if we accept the fact, is, as I have said, no proof at all. It is partly that in some cases in which a direct pyramidal tract has been found, there has been weakness of body or increase of reflexes on the non-hemiplegic side. But this proves nothing either, for the affection of the non-hemiplegic side of the body often occurs without any abnormality being perceptible on that side of the cord.

The proof of the variation is then of the most tenuous character, and bearing in mind the objections to it on general grounds, we may safely regard it as purely hypothetical.

And we may go further, and say that in the present state of our knowledge there is little justification for believing that the pyramid fibres end on both sides of the cord. For in certain cases of hemiplegia from cortical lesion, no clinical symptoms can be observed on the non-hemiplegic side, so that we must conclude that the pyramid fibres end on one side only of the cord. When clinical symptoms are observed on the non-hemiplegic side, they are in all probability due to an alteration of the grey substance, brought about by the degeneration of the pyramid fibres.

The Posterior Columns.—Our knowledge about the direct continuation of nerve-fibres of the posterior roots of the spinal nerves into the medulla, we owe chiefly to the observation of Bouchard[1] (1866), of Lange[2] (1872), of Schultz[3] (1883), on man, and to the experiments of Singer[4] (1881) on dogs. Schiefferdecker[5] (1876) was the first to give a detailed description of ascending degeneration after section of the cord in dogs. From these and other observations we gather the following account of the course of the nerve-fibres of the posterior root. Each posterior root on entering the cord forms the most lateral portion of the posterior column, that next the grey substance; a considerable number of its fibres at once enter the grey substance, the rest continue upwards, becoming, as they go, more mesially situated, in consequence of the entrance of other nerve-roots, and giving off, in their course, fibres to the grey substance of the cord. But not all the nerve-fibres end in the cord; some run on to end in the clava; the course of these fibres has been worked out for the sacral and lumbar nerves only. From what has been said, it is evident that the several posterior nerve-roots form ascending laminæ of fibres, so that in the upper lumbar region the nerve-root which has last entered forms a lamina in contact with the posterior cornu, and each nerve-root below this forms a lamina immediately mesial to that of the one above it, the meso-dorsal angle of the posterior column being occupied by fibres from the last sacral nerve. As these laminæ ascend in the cord, they give off at intervals, and chiefly in the dorsal region, a considerable number of their fibres to the grey substance of the cord. In the cervical region of the cord all the fibres which have reached it from the lumbar and sacral nerve-roots are found in Goll's column occupying the same relative position as they occupied lower down. These fibres, without much diminution in number, run on to the medulla, and end in the cells of the clava; they form thus an ascending medullary tract.

Hence then, broadly speaking, the nearer the mesial part of the posterior column is to the medulla, the more completely does it become an ascending medullary tract, and the less does it consist of cord fibres simply ascending from the posterior roots. It seems to me probable that it is on this account that in the developing cord—as described by Flechsig—there is in the dorsal cord no area of fibres in which the medulla is developed late, to correspond to Goll's column in the cervical cord. At the same time, one would expect that further observation on the time of development of the medulla of the nerve-fibres would enable the ascending medullary to be distinguished from the ascending spinal fibres in all regions of the cord. Schiefferdecker and Singer with some reservations, and Schultze unreservedly, consider Goll's column to be made up of fibres which come from the sacral, the lumbar and perhaps from the lower dorsal region. The corresponding fibres of the upper part of the cord they apparently consider run in Burdach's column. Since there are ascending medullary fibres in the lower nerve-roots, we may take it as certain that there are similar fibres and of similar function in the upper nerve-roots also. On the above view then, Goll's column contains medullary fibres from the lower part of the body, and Burdach's column contains medullary and spinal fibres from the upper part of the body. The medullary fibres of Goll's column end in the nucleus of the funiculus gracilis; the fibres of similar function in Burdach's column must then end in cells of similar function to those of the nucleus of the funiculus gracilis; thus they either leave the funiculus cuneatus for the funiculus gracilis, and end in its nucleus, or they end in the nucleus of the funiculus cuneatus; in which case the latter—in part, at any rate—has the same function as the nucleus of the funiculus gracilis, the one being the centre for the medullary fibres of the upper part of the body, the other for the medullary fibres of the lower part of the body.

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[1] Bouchard, op. cit. t. vi. p. 463.
[2] Lange, 'Schmidt's Jahrb. d. ges. Medicin,' 1872, p. 281.
[3] Schultz, 'Arch. f. Psychiatrie,' Bd. xiv.
[4] Singer, op. cit.
[5] Schiefferdecker, op. cit. p. 589.

Schultze's opinion is based upon an observation in man in which the middle of the cervical enlargement had been completely destroyed; the degeneration of the posterior columns, complete just above the lesion, was in no part of the cord up to the medulla confined to Goll's column, but occupied also a considerable part of Burdach's column. A similar account is given by Löwenthal of the effect of section of the posterior columns in the cervical region in the dog. These observations are not very conclusive as to the point in question; and it may be noted that no one has obtained complete degeneration of Goll's column in the upper cervical region by section of the cord in the lower dorsal region, and further, that Goll's column certainly increases in its way up the cord. We must be content to wait until observations have been made upon the effects of section of the posterior roots of the spinal nerves in the cervical region.

Since the fibres of Goll's column consist of small nerve-fibres, and since there is ground for believing that medullated nerve-fibres do not alter in size in their course in the cord, we may conclude that the medullary fibres of the posterior columns consist of a greater or less part of the small fibres of the spinal nerves, and that the spinal fibres of the posterior columns (except perhaps in the upper cervical region) include all the large fibres of the spinal nerves.[1]

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[1] According to Gaskell ('Journ. of Physiol.,' vol. vi.; 1885) the *efferent small* nerve-fibres have a different function from the *efferent large* nerve-fibres. Of the function of Goll's column, nothing is known with certainty. It has been supposed to convey impulses giving rise to sense of touch (Schiff), sense of

The question of the presence of commissural fibres in the posterior columns we may omit, since the recent work on the spinal cord does not deal with it.

The Cerebellar Tract.—Singer found on section of the cord in the dog between the dorsal and lumbar regions, that in the mid-dorsal region, besides the degeneration of the tract ordinarily recognised as the cerebellar tract, there were degenerated fibres scattered over the whole of the antero-lateral column, these fibres being fewest at the periphery of the anterior column. In the cervical region the degenerated fibres were much less in number; they were present at the periphery of the lateral column only, but stretched from the anterior to the posterior nerve-roots, although they were much more diffuse ventrally of the ligamentum denticulatum than dorsally of it. These fibres in the cervical region were considered by Singer as belonging to the cerebellar tract.

According to Flechsig, the cerebellar tract in man stretches as a compact bundle of fibres about as far as the ligamentum denticulatum; at its ventral end it becomes diffuse, and scattered cerebellar fibres are found for a short distance in the antero-lateral column. Making the fullest allowance for the scattered fibres mentioned by Flechsig, there still remains a considerable difference between his description for man and that of Singer for the dog. The more recent observations on ascending degeneration, both in man and in the dog, have tended to confirm in the main Singer's account. That in man the ascending degeneration stretches more ventrally than is described by Flechsig has been pointed out by various observers, and especially by Schultze. Löwenthal has observed the ascending degeneration in the dog after section of the cord in the cervical region: according to him the cerebellar tract is fairly compact up to the denticular ligament; beyond this it becomes much more diffuse, but stretches along the periphery of the cord as far as the point of exit of the anterior nerve-roots. Near the denticular ligament it extends some little distance into the lateral column.

The fact, however, that these ventrally placed fibres degenerate in an ascending direction, is alone insufficient to show that they belong to the cerebellar tract. To show this the fibres must be traced up to the cerebellum. According to Löwenthal[1] the ventral fibres do not run in the restiform body as do the dorsal fibres, but run in the lateral region of the pons—probably as the 'aberrirende Seitenstrangbündel' of v. Monakow—twist round the superior peduncle of the cerebellum and thence run to the cerebellum.

Löwenthal, like all previous observers, finds that the degeneration of the cerebellar tract diminishes in its way up the cord;

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temperature, muscular sense. Lately Herzen (Pflüger's 'Archiv,' Bd. 38, p. 93; 1885) suggests that impulses giving rise to sense of touch and sense of cold travel in the posterior columns, whilst impulses giving rise to pain and to sense of warmth travel in the grey substance of the cord.

[1] Löwenthal, 'Bull. Soc. Vaud. Sc. Nat.,' xxi. 92 (1885).

that is, that a considerable part of the 'cerebellar' tract consists of connecting fibres of the cord. Flechsig, on account of the steady increase in size of the cerebellar tract upwards, considered that it received fibres from the cord, but gave none to it; but the fact does not warrant the conclusion, for the tract must increase in size as long as it receives more fibres than it gives off. It may be mentioned that the most of the scattered degenerated fibres—probably connecting fibres—described by Singer in the dorsal region, were not observed by Löwenthal in the cervical.

According to Flechsig, Laura, Pick, and others, the cerebellar tract arises from the cells of Clarke's column. This still requires confirmation. It is somewhat against this connection that the cells of Clarke's column are of medium size, whereas the fibres of the cerebellar tract are large. An objection of a similar nature might be urged against the generally assumed connection of the pyramid fibres with chief process of the "giant cells" of the motor area of the cortex.

Fibres proper to the cord which undergo Descending Degeneration.—These fibres are called by Singer, who restricts the meaning of the term as used by Bouchard, "commissural" fibres. The term is not, I think, a very happy one. In anatomy the word "commissure" has come to mean the connecting portion between bilaterally symmetrical parts; in this sense, fibres which connect the cervical with the lumbar region of the cord do not form a commissure any more than fibres which connect the spinal cord with the cerebellum or with the cerebrum. If "commissure" is used in its wider meaning, then all these fibres are equally commissural, the fibres of the pyramidal tract, and of the cerebellar tract as much as those connecting the different segments of the spinal cord. But it would, I think, be better to restrict the term "commissural." The fibres which degenerate can conveniently be designated according to their connection and their mode of degeneration, whether above or below the point of injury. Thus we have ascending and descending spinal, medullo-spinal, ascending cerebello-spinal, descending cerebro-spinal fibres in the cord. But there are many nerve-fibres in the antero-lateral columns which do not degenerate either above or below the place of section.[1] These might conveniently be called commissural fibres. And the designation would have some justification. The fibres which degenerate have a trophic centre at one end only, and it is very likely that this is due to the nerve-fibres in life conveying impulses in one direction only, for there seems to me no reason to suppose that the fibres of the posterior roots, or the fibres of the pyramidal tracts, ever receive impulses from the spinal cord. On the other hand, the fibres which do not degenerate on section must have a trophic centre at both ends, and in view of what has just been said, it is probable that this is due to their receiving impulse at both ends.[2]

[1] Some of these may be short ascending or descending spinal fibres; the question might perhaps be determined by observing the degeneration in a piece of cord, for example, in the lower dorsal region, when completely severed from the rest.

[2] This is very nearly the view which has been put forward by Schiefferdecker, to account for the absence of degeneration in certain fibres.

If this is so, these fibres are more properly commissural than those which degenerate.

The proper cord-fibres which degenerate in an ascending direction have already been mentioned in connection with the cerebellar tract (p. 105), since the two sets have not as yet been with certainty distinguished.

Of the proper cord-fibres which degenerate in a descending direction, those which lie in, and in the neighbourhood of, the lateral pyramidal tract have also been mentioned (p. 97). The remainder are scattered throughout the ventral part of the antero-lateral column. In the dog the longer fibres form a moderately compact zone at the periphery of the anterior column. The observations of Schiefferdecker[1] and of Singer, from whom chiefly the above statements are drawn, were made after section of the cord in the lower dorsal region. In Löwenthal's experiments the spinal cord was cut through more or less completely in the cervical region; he finds throughout the lower cervical, the dorsal, and the upper lumbar region, as far as the lumbar swelling, a degenerated tract of fibres round the whole periphery of the anterior column like that previously described by Schiefferdecker and by Singer, after section of the dorsal cord. But, unlike previous observers, he finds that this tract, at the junction of the anterior with the lateral column, continues dorsally for some little distance into the lateral column, although leaving the periphery and becoming more diffuse.[2] Since these fibres diminish in number in passing down the cord, they must run to different cord segments. Since, further, the zone in the lumbar region is less compact than that previously described after section of the cord in the dorsal region, we may conclude that it receives from, as well as gives off, fibres to all parts of the cord. Some of them probably arise in the medulla, *i.e.* are descending medullo-spinal fibres.

For the fibres of short course which undergo descending degeneration, Löwenthal finds much the same distribution in the cervical region as that found by Schiefferdecker and by Singer in the lumbar region, viz. in the non peripheral parts of the anterior column, and in "anterior mixed zone" of the lateral column.[3]

Tertiary Degeneration.—Sherrington and myself found that after injury to the brain in the motor area, there was, besides the secondary degeneration in the pyramidal tracts, a degeneration of a different nature, more resembling chronic myelitis. The altered fibres were chiefly in the anterior columns, varying somewhat in position in different parts of the cord. Since this was apparently the result of an alteration in the grey substance of the cord in consequence of the secondary degeneration of the pyramidal tracts, we called it a tertiary degeneration, at the same time pointing out the possibility of its having been produced by the hardening agent. Sherrington has since found two other cases in which, after cortical lesion, a similar change had taken place in the spinal cord. As he points out, the entire absence of altered fibres from the cerebellar tract is strong reason for regarding the "myelitic" appearance of certain fibres in the rest of the cord as being due to degeneration, and not to the hardening agent.

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[1] Schiefferdecker (op. cit. p. 569) considered these fibres to correspond with Türck's 'Hulsenvorderstrangbahn,' in man. They have, however, nothing to do with the pyramids.

[2] These, together with the corresponding fibre of the anterior column, Löwenthal calls "long commissural fibres." If this term is used, it must not be confused with Bouchard's anterior long commissural fibres, which lie in the dorsal part of the lateral column (cf. p. 97).

[3] Schultz (op. cit.) describes in man, after a lesion in the cervical region, degenerated fibres throughout the anterior column and anterior mixed zone; in the mid-dorsal region the degenerated fibres were confined to the inner half of the anterior column.

The Parts of the Cortex of the Brain which are connected with the Lateral Pyramidal Tracts.—It is well known that a lesion which affects a considerable part of the motor area of the cortex of the brain causes a degeneration in the lateral pyramidal tract of the cord, and that a considerable lesion in other parts of the cortex—such as that of the occipital and temporal lobes—causes no degeneration in the cord.

We know very little more than this, but there has been a great tendency to consider this knowledge as much more precise than it really is. This is so because it is commonly assumed: 1st. That the motor area of the cortex, as it has been determined by electrical stimulation, coincides with the "cord area," *i.e.* with the area of the cortex, destruction of which causes degeneration in the cord. 2nd. That each "centre" in the motor area is connected with its corresponding local centre in the spinal cord, and with that only; for instance, that the cortical area, stimulation of which causes movements of the fore-arm, is connected with the "arm centre" in the cervical region of the cord, and with no other part of the cord.

Both of these assumptions follow so naturally from the results of cortical stimulation that it may cause some surprise that I should touch upon them. But it is necessary to do so in order to discuss fairly the results of cortical extirpation.

Ferrier and Yeo,[1] in a very interesting paper on the physiological effects of extirpation of the cortex in the ape, touch somewhat incidentally on the question of secondary degeneration. In the cases in which the cord was examined, secondary degeneration was only found when the cortical lesion had taken place in the motor area (the convolutions bounding the fissure of Rolando). These observations are distinctly in favour of the view, that the "cord area" coincides with the motor area for the limbs and trunk, but the details given are not sufficiently full to prove that this is the case.

The more recent experiments in the dog are difficult to harmonise with the scheme given above.

Sherrington and myself, from a comparison of the area of greatest degeneration on the two sides of the cord consequent on destruction of the cortex of the brain to unequal extents and at different times, were inclined to believe that the anterior part of the motor area was connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the anterior part of the lateral pyramidal tract; and further, that the destruction of the cortex behind the motor area as figured by Hitzig and by Ferrier would give rise to degeneration in the cord. The nature of our observations did not, however, allow of conclusions being drawn with certainty. It will be noticed that our tentative conclusions involved: (1) That fibres from each "centre" of the motor area run in a fairly compact bundle. (2) That the cord received nerve-fibres from parts of the cortex which are considered to be motor centres for certain of the muscles of the head. (3) That the "cord area" stretches farther posteriorly than the described motor area.

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[1] Ferrier and Yeo, 'Phil. Trans. Roy. Soc.,' pt. ii., 1884, p. 479.

The posterior part of the cord area—as given above—lies in part, at any rate, in the region which, in Ferrier's observations, produced, on stimulation, movements of the eye. Ferrier considered it, however, to be a part of the "sensory" area of the cortex. This region is called the "Fühlsphäre" for the eye by Munk, who puts it in the same class with the cortex of the sigmoid gyrus, which he calls the "Fühlsphäre" for the limbs and neck.

Löwenthal's observations deal with these questions. He has examined the brain and cord of a considerable number of dogs from which various parts of the cortex had been removed by Schiff. Unfortunately the lesions were in too many instances insufficiently localised.

Löwenthal has observed five cases in which the coronal and anterior ecto-sylvian convolutions—the anterior parts of the 3rd and 2nd convolutions—were largely removed; in two of these cases a part also of the median ecto-sylvian and of the anterior sylvian convolution were also removed. In each case, however, a small part of the postero-lateral edge of the sigmoid gyrus was affected; in one case this appears to have been a mere fraying of the edge of the sigmoid gyrus. In every instance there was a degeneration of the lateral pyramidal tract of the spinal cord, a degeneration which could be followed a variable distance in the dorsal region.

If the degeneration was due to the destruction of the coronal, ecto-sylvian or sylvian convolution, it follows that the destruction of the motor centres for certain muscles of the head, causes degeneration of part of the lateral pyramidal tract of the cord. It is unlikely that the slight and superficial injury to the sigmoid gyrus should have caused so considerable a degeneration as occurred in these cases; but even by granting this, the difficulty is not removed, for the part of the sigmoid gyrus injured[1] is the motor area for the fore-limb, and the destruction of this ought not, on the generally accepted view, to cause degeneration in the dorsal region of the cord.

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[1] Paneth, in his recent paper, places one of his small motor areas for the hind-limb near this spot.

It may be, however, that in each of these cases the lesion was carried too deep, and affected part of the corona radiata containing fibres coming from the cortical "centre" for the hind-limb. Löwenthal mentions this possibility, but does not say that there was any evidence for it in the sections of the brain.

Sherrington gives an observation which is more difficult to explain on the ordinary view of the connection of the motor centres with the cord. He finds that a lesion which does not reach farther forward than the posterior edge of the ecto-sylvian fissure, produces degeneration throughout the cord, *i.e.* a lesion which lies posteriorly to the motor area as given by Hitzig, Fritsch, and by Ferrier,[1]—though including part of the "Fühlsphäre" of the eye of Munk—causes degeneration in the lateral pyramidal tract. In his case it is very difficult to see how fibres from the sigmoid gyrus could have been affected.

Further, Löwenthal finds that lesion of the anterior limb of the sigmoid gyrus—the posterior lateral border being left intact—leads to degeneration as far as the upper dorsal region.[2] No one, I believe, has considered the anterior limb of the sigmoid gyrus to contain a motor area for any of the muscles of the trunk or of the hind-leg.

From the destruction of certain parts of the cortex, Löwenthal has observed no degeneration in the cord. In a case in which the lesion was confined to the postero-dorsal part of the sub-orbital lobe degeneration was absent. Sherrington also found no degeneration from injury of the sub-orbital lobe. According to Munk, the sub-orbital lobe is the "Fühlsphäre" of the trunk.[3]

In another case described by Löwenthal, in which degeneration was absent, a roundish area of the cortex was destroyed, involving the mid-third about of the ectolateral convolution, by passing a short distance over the lateral fissure into the fourth convolution, and nearly reaching the supra-sylvian fissure. In a third case the roundish area destroyed was a little larger than the preceding; cutting across the median ectolateral (2nd) convolution, it just passed over the supra-sylvian fissure dorsally, and nearly reached the sylvian fissure ventrally; a prolongation of the sylvian fissure would have nearly bisected the area of the lesion.

The tentative conclusion mentioned above, that the fibres from different parts of the motor area run in different parts of the lateral pyramidal tract, has received no confirmation. Löwenthal examined the cord in seventeen cases of cortical lesion affecting the sigmoid gyrus in different degrees and in different regions. He mentions no difference in the cord degeneration, except a difference in intensity. Sherrington finds the position of the cord degeneration to be the same whatever the cortical lesion which has given rise to it. If this is so, it shows a very remarkable intermingling of the fibres of the pyramidal tract in their course from the cortex.

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[1] Luciani and Tamburini also place the motor areas for the trunk and limbs in the sigmoid gyrus.

[2] The lower dorsal and lumbar regions were not observed.

[3] Ferrier ('Transact. Royal Soc.' 1884, p. 528) mentions that in the ape, destruction of the frontal lobes anterior to the motor region, causes degeneration of the mesial part of the crusta, but none in the pyramid.

A limited intermingling of fibres is suggested by the observations of Hitzig, who found that movements of the muscles of the trunk could be obtained by stimulation of the motor area for the fore-limbs; and a greater intermingling is suggested by the recent observations of Paneth[1] made under Exner's direction. Paneth finds that there are small motor areas for muscles of the fore- and of the hind-limbs interspersed over a considerable area of the posterior limb of the sigmoid gyrus.

But both as regards this point, and as regards the connection of the part of the motor area with the lateral pyramidal tract, we must wait for further investigations. The experiments which have been mentioned above suggest conclusions which are not in accordance with those which we should on general grounds be inclined to accept; for this reason, then, we are justified in reserving a definite opinion until fuller and more satisfactory evidence is forthcoming.

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[1] Paneth, 'Pflüger's Arch.,' Bd. 37, p. 521 (Dec. 8, 1885).—See Abstract in the present number of 'BRAIN,' p. 144.

ON SOME RECENT ADVANCES IN OUR KNOWLEDGE OF THOMSEN'S DISEASE.

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(1.) 'A Consideration of the Thomsen Symptom complex, with reference to a new form of Paralysis Agitans.' By ALLAN McLANE HAMILTON, M.D. *The Medical Record*, New York, January 23, 1886.

(2.) 'Klinisches und Pathologisch-Anatomisches von der Thomsen'schen Krankheit.' Von Prof. W. ERB. *Neurologisches Centralblatt*, No. 13, 1885.

(3.) 'Thomsen'schen Krankheit bei vier Geschwistern.' Von Prof. EULENBURG und Dr. MELCHERT. *Berliner Klinische Wochenschrift*, No. 38, 1885.

(4.) 'Une Observation de Maladie de Thomsen.' Par MM. PITRES et DALLEDET. *Archives de Neurologie*, No. 29, 1885.

(5.) 'Ein Fall von Thomsen'schen Krankheit.' Von DR. G. FISCHER. *Neurologisches Centralblatt*, 15th February, 1886.

(6.) 'Maladie de Thomsen et Paralysie pseudohypotrophique.' Par VIGOUROUX. *Arch. de Neurologie*, vol. 8, No. 24, Nov. 1884.

(7.) 'Observation d'un cas de Maladie de Thomsen.' Par L. DELIGNY. *L'Union Médicale*, 1885, No. 5.

(8.) 'La Maladie de Thomsen (Dysmyotonie Congénitale).' Par LONGUET. *L'Union Médicale*, 1883, November 25.

(9.) 'Contribution à l'histoire de la Maladie de Thomsen.' Par P. MARIE. *Revue de Médecine*, 1883, p. 1064.

(10.) 'Fem Tilfälde af den Thomsen'ske Sygdom. Af KNUD PONTOPPIDAN' (*Hosp.-Tidende*, 1884, 3 R. II., 34).

(11.) 'Ueber eine dem Bilde der cerebrospinalen grauen Degeneration ähnliche Erkrankung des centralen Nervensystems ohne anatomischen Befund, nebst einigen Bemerkungen über Paradoxe Contraction.' WESTPHAL, *Archiv für Psych.*, Bd. XIV., p. 87 (especially p. 130).

(12.) 'Malattia di Thomsen. Sua Analogia coi fenomeni muscolari dell' ipnotismo,' Sepilli, *Arch. Ital. per le malattie nervose ec.* Fasc v. 1885.

(13.) 'La Malattia di Thomsen.' PETRONE, *Lo Sperimentale.* July, 1884.

(14.) 'Handbuch der Kinderkrankheiten.' GERHARDT, t. v., p. 175.

(15.) 'A Case of Thomsen's Disease; a form of Paresis of Motion, accompanied by muscular hypertrophy.' ENGEL, *Philadelphia Medical Times*, September 3, 1883.

(16.) 'Thomsen's Disease.' By PAUL CHAPMAN, M.D. *Brain*, vol. vi.

In the article last mentioned there is an almost complete list of the literature of the subject up to that date, and I think the fifteen other references that I have enumerated made the whole of the literature very nearly, if not quite complete up to the present date. The first five of the above articles will be discussed in detail, for not only are they the most recent, but also the most important. The sixth article does not call for comment, and the seventh relates an example of the disease in which the patient was a man 25 years old; his father had had the disease, but his general health had in no way been affected by it. The patient was the only child; the cramps caused great pain, and came on several times a day, and lasted a long while; the reflexes were normal. The peculiarity of the case seems to be that the contractions lasted so long, and there was so much pain with them.

The eighth article gives a very good account of the literature of the subject up to date, but makes confusion worse confounded by using another term, viz. dysmyotonie congénitale, almost adopting Strumpell's term, congenital myotonia. The exact nature of the case in the ninth article is probably rather doubtful, for the cramps were strictly one-sided, and the patient also suffered from epileptiform seizures. The tenth article records five cases, two in one family, and three in another. Portions of muscle were examined microscopically, and are stated to have been healthy. This paper is to be found abstracted in the 'Neurologisches Centralblatt,' 1884, p. 520.

In the eleventh of the above-mentioned papers Westphal discusses briefly the nature of paradoxical contraction, and comes to the conclusion, that the contraction of Thomsen's disease is allied to it, but that our knowledge about both of them is at present very elementary.

Petrone's account of the disease is good, brief, and to the point, but does not contain anything new about it.

The first of the above articles begins with a historical account of the malady. It is pointed out, as is well known, that Sir Charles Bell, in 1836, mentioned the existence of a disease which was probably the same as the one now under discussion, but gave it no distinct name. The next person to call attention to it was Thomsen, who in 1876 described the disease as occurring in members of his own family. Since that time many examples of the disease have been recorded, chiefly in Germany, together with a few in each of France, Sweden, and Italy, and two in America. Altogether about twenty to thirty cases have been recorded. Hamilton gives the clinical expressions of the disorder as follow:—(1) a sudden spasmodic muscular seizure which attends the performance of a voluntary act; (2) its subsidence after the repetition of the act; (3) an apparent increase in bulk in several muscles which are chiefly and most frequently the subjects of cramp; (4) an increase in electro-muscular irritability; (5) a normal or slightly excited state of tendinous reflex activity; (6) these symptoms are often of congenital origin, and progressive as regards intensity.

I shall show later on that all these symptoms are in the main correct, but that by the light of our present knowledge they can be much more definitely described. Hamilton points out that the lower extremities are most often implicated, and that hence when the patient essays to walk, there is sudden flexion of the thigh and leg, in which position, owing to the stiffness in contraction of the muscles, they remain in this condition from five to ten seconds, so that the patient often falls. It may be some time after a volitional impulse that the muscle contracts, when it does there is this stiffness [Muskelssteifigkeit]. The muscles of the face may be affected. Cold and mental excitement increase the liability to the disease. In only one case were the reflexes exaggerated; a congenital history is not always the rule. Hamilton's object in writing the paper is to throw doubt upon the question whether Thomsen's disease is to be looked upon as a distinct malady, for he points out that this muscle-stiffness on contraction is a frequent symptom in a variety of maladies; in most cases it is purely psychic; it is an occasional symptom of the third stage of locomotor ataxy; it is common in hystero-organic disorders of the nervous system, and may be associated with coarse cerebral lesions, or may be present in Parkinson's disease. No mention is made of the recent article by Erb and others, or of the microscopical appearance of the muscles, nor are the electrical reactions and mechanical irritability reactions referred to in much detail; and although the question is still under discussion, and awaiting a promised article by Erb on the subject, I think we shall see that whilst it is by no means deniable that cramps may take place in a number of diseases, still this group of symptoms taken with the changes in the muscle, points strongly to the fact, that Thomsen's disease must be looked upon as a genuine and distinct malady. In further proof of this it may be observed that the hitherto recorded cases have not been suffering from paralysis agitans, nor any other disease mentioned by Hamilton. The author then gives an account of a boy, aged 10 years, who had always suffered from cramps, which were worse under mental excitement, and caused him to walk in a squat position. No change could be detected in the portions of muscle removed, and the electrical reactions were normal. Both these facts are against the case being one of Thomsen's disease, and no diagnosis seems to have been made. Another case is given in which a boy, 9 years old, who had suffered from headache and attacks of vomiting, had muscle-stiffness on contraction in the voluntary muscles; muscular enlargement and loss of power; the mouth was drawn slightly to the left side; the tongue pointed to the right; there was a prominence of the eyes and a slight lateral nystagmus. The headache and vomiting do not necessarily preclude Thomsen's disease, as the cases of Eulenburg and Melchert show; still the other symptoms probably do, and so we have here another example of cramps occurring in other diseases. For the convenience of those who wish to look up the subject, I may mention that Thomsen's original article is to be found in the 'Archiv für Psychiatrie und Nervenkrankheiten,' 1876. Hamilton, at the heading of his article, uses the phrase "Thomsen symptom complex." This is a pity, for not only does it lead to confusion, because the phrase "symptom complex" has a wider significance, as we see in an article by Westphal, "Ueber einen eigenthumlichen Symptomencomplex bei Erkrankung der Hinterstränge des Rückenmarks,"[1] where the "Symptomencomplexes" apparently included paralysis, paradoxical contraction, rigidity and cramp in certain movements; but a much better phrase, viz., myotonic contraction, has been proposed, which is shorter and less cumbrous than "Thomsen symptom complex."

Erb begins his article by pointing out, that although there has never been a post-mortem examination on a case of Thomsen's disease, yet the five observers who have excised portions of the muscle have failed hitherto to find any change in it. He gives an account of two brothers, aged respectively 14 and 15 years, who had it typically, and in whom it had been present some time. The voluntary power of the muscles was slightly diminished, especially considering their size. On movement of the voluntary muscles there was the usual trouble, viz. a slowness and difficulty of contraction; then a stiffness and tension, which suddenly passed off; and then there was free movement. With a very powerful voluntary movement the duration of this tonus was from 10–25 seconds. The motor nerves with mechanical stimulation showed diminished excitability. With the faradic and galvanic currents the reactions of the nerves are practically normal, unless the stimulations follow very quickly, when there is a slight lasting contraction. In the muscles the mechanical excitability is increased, and after a strong stimulus remains from 12 to 30 seconds. A strong faradic current gives a contraction which remains for 20 or more seconds; but on opening, there is only a momentary contraction with even the strongest current. With the galvanic current the muscles usually give only closing contractions. K and A are usually alike, but in some muscles Ka S Z may be best, in others An S Z. It is with Ka S Z that one gets the best tonic contraction, which is often extremely good, and lasts 5–30 seconds. With the stimulations rhythmically following one another, we get wave like contractions also following each other, and going along the muscle from the kathode to the anode. This is well seen in the flexors of the fingers, the vastus internus and externus. All the above reactions are best shown in muscles which are under the influence of the will. The essentials are the slowness and duration of the contraction; a good name for it is myotonic contraction or myotonic disturbance of muscles. It shows itself on direct stimulation of the muscle, or, in the case of the nerve, when one stimulus quickly follows another.

On microscopical examination of a piece of excised muscle in the fresh state, the only thing that could be made out was that the fibres were a little broader than natural; but with Müller's fluid preparations they appeared many times wider than they should be, and not polygonal, but rounded. The interstitial tissue and vessels were normal; there was considerable thickening of the sarcolemma.

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[1] 'Archiv für Psych.,' etc., 1885, Bd. xvi.

Eulenburg and Melchert have given a most interesting account in which the disease affected four brothers and sisters. The family history was unimportant; the mother suffered from cramp in the stomach and headache. Of the six children, all suffered from cramps and convulsions during teething, and during their school-days from hemicrania and vomiting. One daughter had migraine; another used to wake up with attacks of cardiac pain and palpitation. The three sons and the eldest daughter were typical examples of Thomsen's disease. In all of them the voluntary muscles were affected, and there was the usual contraction. The following are the details of each of the four cases.

W. K., 22 years old; he was first attacked when 7 years old, and then he found stiffness on attempting to run; this continued off and on, and from 14 to 18 he suffered very badly, so that he would often fall in the street; after 18 he became a little better; but during his service in the army the disease gave rise to so much trouble, that he had great difficulty in carrying out correctly the exercise orders; in the same way it was difficult, in shooting, to raise the musket to the shoulder, and he had much difficulty during his apprenticeship as a dentist. On examination, the peculiarities of contraction of voluntary muscles, as described in the two previous articles, were most apparent, and, as already mentioned, after a few contractions the myotatic contractions passed off. The peculiarity was present in the forearms, lowers, body, face, deglutition muscles, tongue and eye muscles. The myotatic contraction was most marked the longer the muscles had been at rest, worse after cold and inanition, but better after meals. On direct mechanical stimulation, after a short time the myotatic contraction appeared, lasting 3–5 seconds. With faradic stimulus to muscle and nerve there was nothing noteworthy. With the galvanic current to the muscles, K S Z appeared sooner than A S Z and A O Z > K O Z, but the contractions came slowly, and lasted 2–5 seconds; with galvanic current to the nerve, K S Z appeared sooner than A S Z, but the contraction was momentary. Patellar reflex weak.

George K., 21 years old; as a child had headache and vomiting. When a boy, had slight difficulty of movement, which showed itself in gymnastics. His muscles were, when he performed voluntary acts, in the same myotatic condition as those of his brother, but worse. Mechanical stimulation to muscles gave the same results, but they were more pronounced. Faradism as in brother; galvanism to nerve K S Z > A S Z, with no myotonic contraction; this and muscular galvanism also being the same as the brother. There was a prolonged latent period; there was in succession a prolonged contraction, and then a sudden relaxation, best seen after prolonged rest; frequent slight tremors of the muscles were to be seen, and at times there were very strong opening contractions with the kathode.

Carl K., 21 years old. Headache and vomiting when young. The trouble with the muscles came on at 12 years, when he was found to be bad at gymnastics. The symptoms were not so marked in his case; the mechanical myotatic contraction was present in slight degree. Faradism gave no lasting contractions; the galvanism results were just the same, but weaker. Patellar reflex weak.

Louise A., 29 years old. The disease first appeared in childhood, and got worse at puberty. She has great difficulty in going upstairs, and in executing complicated movements, as dancing. All the symptoms were pretty much the same as in the brothers. The author directs attention to the fact, that the two children who have not had Thomsen's disease have anomalies of their nervous system; also that the symptoms increase after cold and exhaustion, and the daughter was better when she lived in more comfortable circumstances. The four cases showed slight qualitative differences to their electrical reactions, and Erb's wave contractions with strong current were not observed, but there was a tendency to tremulous oscillations with faradic stimulation.

MM. A. Pitres et Dalledet have given a very full account of a patient suffering from this disease; they also give several myographic tracings. The grandparents and the father were healthy. The mother at the age of 15 suffered from muscular stiffness, like that which her son, the patient, suffers from now. This difficulty disappeared in two or three years; then there were cramps, chiefly in the sacro-lumbar region, the pains coming on day or night, without any relation to movement. She and her husband had six children; the three first being like the father in all points, the three younger, on the contrary, suffering from muscular stiffness on voluntary movement. Of these, the fifth is the patient, and will be described in detail; the fourth, a man of 26 years, is exactly like the patient in all the symptoms, but not so bad; and the sixth, a girl of 19 years, had a difficulty of voluntary movements when 15 and 16, but that has now disappeared. The difficulty of voluntary movement of which the patient complains began when he was a child. Up to the age of 22 it increased but slowly; since then it has gone on very quickly. He is now 24.

The first thing that strikes one is the hypertrophy of the muscles, which is well seen in the arm and forearm, and the thenar and hyperthenar eminences. The sterno-mastoids, triceps of thigh, and sacro-lumbar muscles are all hypertrophied. During contraction they are irregularly bossed, and as hard as wood. The power of the muscles is not so great as their hypertrophy would lead one to expect. The electrical reactions are normal. The difficulty of voluntary movement consisted in a restraint and stiffness on the attempt, which gradually passed off as the movement was frequently repeated. This is very well shown by a myographic tracing, which shows that the latent period is increased considerably, and the muscle remains contracted much longer than it should. The authors also insist on a point which their tracings show, but which has not been noted in other cases, namely, that not only is there a difficulty of contraction, but also of the first *décontractions*; in fact, in the tracings the down-stroke often extends over a longer period than either the up-stroke or the crest. With a strong and rapidly repeated faradic current, the same thing is observed in the early contractions. This, it will be observed, brings this case somewhat in harmony with Erb's electrical myotatic contractions, and perhaps when the authors here speak of the electrical reactions normal, they did not look for the myotatic reactions. The peculiarities of contraction were increased by cold. The muscles of the face would sometimes remain in an emotional expression so long, that the hand had to be used to aid their return to normal, hence, sometimes there was difficulty of speech. The fear of the peculiarity being observed increased it. The tongue, deglutition, phonation, respiration and ocular muscles were not affected. Passive movements were easy, and sensibility was intact; reflexes normal, sensation of position in space normal. All other functions of body healthy.

The fifth paper, mentioned at the beginning of this notice, has only just been published. The author observes that in this case the skin and tendon reflexes were normal, and that the strength of the muscles was not so much increased as would have been expected from the increase of volume. Sometimes the myotonic contraction remained from 25–27 seconds after voluntary contraction, but after repeated movements it disappeared. On the whole, this case agrees with others, and hardly differs more from them than they do among themselves. The myotatic reaction, in response to direct muscle stimulation, was not so easily obtained, and no K O Z could be obtained on stimulation of the nerve. The rhythmic wave-contractions of Erb were shown very well indeed, and with a current of a strength of 15–20 M A, they were well formed, slow, and about 15–20 seconds apart, passing from the kathode and the anode. With a strong faradic current there were rapid oscillations of the muscles. On microscopic examination of the fibres they are found to be thickened. In no parts was the cross striping well marked. The contour of the bundles is uneven, and has here and there little tumefaction. The author raises the question whether these appearances are due to the mechanical stimulation of excision.

In conclusion, we may, I think, gather that the following are the chief characteristics of Thomsen's disease:

1. It occurs in young subjects, coming on usually in youth or childhood.
2. It lasts a long while; is never fatal; and may pass off.
3. The muscular difficulty is much increased by cold and mental excitement.
4. It occurs in members of the same family, and is slightly hereditary.
5. Oth r neurotic disturbances in the family are by no means always well marked.
6. Voluntary muscles only are affected, the legs perhaps oftenest.
7. There is an increase of bulk in the muscles.

8. But considering their increase in size, they are not so powerful as might be expected.

9. On attempted voluntary movement the latent period is usually prolonged; then comes a severe contraction, lasting from five to thirty seconds; then in some cases a sudden, and in others a slow relaxation. This peculiarity constitutes a myotonic contraction.

10. The above is more marked, the longer has been the interval of rest. After repeated contractions the difficulty becomes less, and soon passes off, the contractions being ordinary.

11. Mechanical stimulation of nerves shows a diminished excitability. Faradic or galvanic stimulation of nerves gives slight myotonic contractions on being rapidly repeated.

12. Mechanical stimulation of muscles shows increased excitability, with long myotonic contractions. Galvanic stimulation usually gives closing contractions; K S Z is generally the best, giving a good myotonic contraction, but the exact order is variable. Strong faradic current on closing gives a good myotonic contraction; on opening, a very slight contraction.

13. With stimulations rhythmically following each other, we get wave-like contractions passing along the muscle from the kathode to the anode.

14. The reflexes are normal.

15. There is increased width of the muscle fibres, which lose some of their striation, are rounded instead of polygonal, and have protuberances on their contour. The sarcolemma is thick.

Reviews and Notices of Books.

I. *Beiträge zur Anatomie und Physiologie.* Von Prof. C. Eckhard (Giessen). Vol. X. No. IV. *Geschichte der Experimental Physiologie des Froschhirnes.*—4to. Giessen: Emil Roth, 1883.

II. *Untersuchungen über die Physiologie des Froschhirnes.* Von Dr. J. Steiner (Heidelberg). 1 vol. 8vo. pp. 143. Braunschweig: Vieweg, 1885.

III. *Saggio Sperimentale sul meccanismo dei movimenti voluntari nella Testuggine palustre.* Del Prof. Giuolio Fano. 1 vol. large 8vo., pp. 61. Firenze: Lemonnier, 1884. *Sul nodo Deambulatorio Bulbare.* Del Prof. G. Fano. 1 vol. pp. 23. Genova, 1885.

In the second half of the 17th century, Robert Boyle in England, and Francesco Redi in Italy, performed the first experiments on record, with a view to determine the functions of the brain in amphibians and reptiles. In his essay on "The Usefulness of Experimental Natural Philosophy," published in 1663, the former appears to be conscious of the fact, that the ablation of the frog's brain abolishes spontaneous movements, whilst the latter observed a similar phenomenon, though not so constantly, after excerebration of tortoises. Other early observers repeated the experiment without adding materially to these results. Humboldt (1797) appears to have been the first in the application of galvanic stimuli to the cerebral centres, but without obtaining any motor effects.

Rolando (1809) found that integrity of the medulla was a necessary condition for the keeping alive of cold-blooded animals after complete excerebration; and that after removal of the hemispheres only, animals moved fairly well on external excitation. The cerebellum he thought to be an organ of locomotion, comparing its lamellated structure to the recently discovered voltaic pile. Three years later Legallois published his 'Expériences sur le principe de la vie,' in which we find some criticisms upon the imperfect methods followed by his predecessors. He introduced the method of successive sections across the encephalon, and so was able to determine the seat of the respiratory centre in the medulla. Legallois made numerous experiments on the cerebral innervation of the heart, a subject still more fully worked out in Wilson Philipp's papers, published in the 'Philosophical Transactions' for 1815.

It was Flourens who first attempted to define the functions of the several divisions of the encephalon. The first edition of the 'Recherches expérimentales sur les propriétés et fonctions du système nerveux dans les animaux vertébrés" appeared in 1824. He found that the ablation of both hemispheres was necessary to deprive the frog of its motor spontaneity and its sensibility; but afterwards he qualified the latter statement by distinguishing between "sensation" and "the perception of sensations, or intelligence." He maintained that rotatory movements follow removals of the optic thalamus and optic lobe; in the first case they are towards the side of the lesion, in the second in the opposite direction. His well-known discovery of the coördinative function of the cerebellum was not made on the frog, in which this organ is rudimentary. Coster, a pupil of Rolando whose name Flourens does not mention, made a violent attack upon the latter's originality. Though Flourens never denied having read the works of his predecessor, Magendie had no difficulty in showing the wide difference existing between the methods and results of the two observers.

Magendie, Desmoulins, and Serres, confirmed, whilst they partially corrected and extended, the conclusions reached by Flourens. Paton, in a paper published in the 'Edinburgh Medical Journal' (1846), described for the first time the phenomenon since known as the Goltz's "Quackversuch," and which consists in eliciting a croak from a frog deprived of its hemispheres, by stroking it over the interscapular region. Other observers, such as Cayrade (1864), studied the swimming movements of the similarly prepared animal, and the question whether it could "see" gave rise to many discussions, in which, among others, Renzi (1863), Goltz (1869), Vulpian (1866), and Blaschko (1880), took part. It was shown that the hemispheres are not necessary to enable the frog to avoid, or jump over, obstacles; but how far these actions are conscious, and not merely coördinated and adapted visual reflexes, is a problem to be settled only in the light of more extensive researches in comparative psychology. The same remark applies to other reflex phenomena, where the excitation is conveyed through the auditory or tactile organs, and which are found not to be abolished by the ablation of the hemispheres.

Dr. Steiner's book constitutes the most recent contribution to the subject now before us, and is a model of what a work of its kind should be. In the introductory chapter we find a clear statement of the conditions under which experiments on the frog's brain are to be conducted. The author insists upon the necessity of giving the animal perfect rest after the operation, and of then examining its behaviour both on land and in water. He speaks of the importance of accuracy in producing the lesions, and in defining them post-mortem, and accepts Goltz's expression of "Ausfallserscheinung" to denote the symptoms due to the absence of a portion of the brain, in opposition to that of "Nebenwirkung," or "Reizungserscheinung," which is applied to the effects of the traumatism—terms which could be rendered in English by "direct," or "essential," and "indirect," or "concomitant," respectively—a distinction equivalent to that made between destructive and irritative lesions in human pathology.

Dr. Steiner has studied carefully the conditions under which the frog, deprived of its *hemispheres*, can be made to jump over an obstacle, instead of avoiding it, and proves the importance of visual impressions in the execution of these acts. Goltz's "Balancirversuch" is fully discussed. When the animal is placed upon a small board, which is gradually tilted upwards, it begins to ascend, and finally rests upon the edge. On continuing the movement, and so lowering the board in the opposite direction, the frog begins to descend, raising its head, instead of depressing it, as during the ascent. These phenomena are purely reflex, and depend, as Dr. Steiner shows, upon different impressions, due to the strain upon the muscles and joints of the head and neck, arising from altered equilibrational conditions.

Careful observation of the behaviour of the prepared frog in water, proves that the influence of vision has hitherto been insufficiently taken into account. Placed in a quadrangular basin with perpendicular walls, the animal makes for one of the corners; then directing its eyes upwards, takes a spring that lands it on the edge, where it assumes the characteristic motionless attitude. Here, as in the experiments with obstacles, conditions of light and height are necessary to obtain these results; the frog does not attempt to jump unless the light shines from above, and the top is not too high to be reached.

The effects of a removal of the *thalami* (in addition to that of the hemispheres) does not seem to have been thoroughly investigated before Steiner. Within two or three hours the skin, even when of the lightest yellow, turns to a dark-brown tinge, which persists till the death of the animal. The movements, both on land and in water, which ensue from peripheral excitations, are somewhat clumsier, and there is no ascent of the inclined plane, the head alone executing the customary motion. The frog, however, still turns over when placed on its back. Unless the optic nerves have been divided at the same time, removal of the thalami does not appear to exert any influence upon the visual reflexes, and the organs appear to be connected with musculo-articular, rather than with cutaneous feelings.

The *optic lobes* of the frog are the homologues of the mid-brain of the higher vertebrates, and consist of two rounded masses containing a ventricular cavity. We must distinguish between their upper and lower parts, which we may designate as "roof" and "base" respectively. The roof represents the corpora quadrigemina in man, and Renzi (1863) has shown it to be necessary to vision. Cayrade's experiments tended to show that the optic lobes of the frog have functions similar to those of the cerebellum in the higher animals; while those of Goltz demonstrated that whereas certain reflexes (Quack- and Balancir-versuch) disappear with the ablation of these organs, others (turning-over, reaction to cutaneous excitations) still persist. Newell Martin (1878) states that under the same circumstances the respiratory movements are diminished, but become tetanic on excitation.

Dr. Steiner shows that the utmost care is required to obtain trustworthy results from the ablation of the optic lobes. A frog so prepared still jumps fairly when touched, but has lost some of its swimming powers. In their normal condition frogs swim in two different ways; in the first, or fully coördinated mode, they hold the fore-legs adducted and drawn backwards along the side of the body, whilst the hind-legs perform energetic movements of flexion and extension; in the second, the fore-legs are kept semi-flexed in front, the hind-legs being used in a more leisurely, paddling way. Now ablation of the lobes reduces the swimming powers of the animal to the second mode.

When the anterior two-thirds only of the optic lobes are removed, a curious phenomenon is observed; on exciting the frog in the usual way (*i.e.* pinching the toes of the hind-leg), it begins to walk backwards. Dr. Steiner very justly remarks that this does not imply the existence of a special centre for that movement, but merely a disturbance of the afferent part of the mechanism upon which the coördinated reflex depends. The same remark applies to the altered mode of natation, which is observed likewise on partial ablation of the lobes.

It is very probable that the effects ascribed by previous observers to a removal of the frog's *cerebellum*, were due to inaccuracies in the performance of the operation. When the latter is strictly limited to the part, none but slight disturbances of movement ensue. The animal performs the usual ascent of the slanting board, but on its making efforts, a certain unsteadiness and tremor is noticeable. When it jumps out of the water upon the edge of the basin, the spring is not accurately measured, being either too long or too short. Removal of the cerebellum from an animal already deprived of its optic lobes, gives rise to no additional symptoms, a fact not surprising when we consider the small size of the organ.

It thus appears that a frog deprived of every part of its encephalon except the medulla oblongata, is able to preserve its natural posture when at rest, to jump, to swim, to right itself when placed on the back. It is true that earlier observers (Rolando, Renzi, &c.) went further, and thought the animal was still conscious of sensations; but we know how fallacious are all such conclusions drawn in the lower animals from apparently purposive reflex movements. Now the localisation of those motor mechanisms is in the anterior part (pars commissuralis) of the bulb which lies under the lamella-like cerebellum; for when a section is made at the level with the posterior edge of the cerebellum, the frog remains limp, whether on land or in water, and no longer reacts to excitations by coördinated locomotor movements. When placed on a rotating disk, it no longer turns its head to one side, as is otherwise the case, nor does it turn round when placed in the dorsal position. The commissural portion of the bulb must contain motor centres for the muscles of the head, limbs, and trunk, which are brought into play by peripheral excitations and by disturbance of the equilibrium of position. Dr. Steiner further shows that these centres are in direct organic relationship to one another, and do not require the intervention of any higher centre to bring them into harmonious play. This fact leads him to consider the influence of the bases of the optic lobes on locomotion, and he concludes to the sensory nature of their function. Experiments are adduced to show that after extirpation of the optic lobe on one side, cutaneous excitations on the opposite side of the body are not followed by a jump, as they are on the other. It would thus appear that in the frog all the motor cerebral mechanisms are in the bulb, and that the other encephalic organs serve only for the elaboration and coördination of the afferent impulses on which spontaneity and psychical life depend.

Dr. Steiner distinguishes between the simple "excitomotor" reflexes of the cord, with the more complicated processes of the bulbar general motor centres and which he terms "sensorimotor" without wishing to imply that they are above the plane of consciousness. A frog deprived of all its encephalon beyond the medulla oblongata reacts by a jump to the application of a weak acid to the skin. When the solution is stronger, the locomotor reflex is succeeded by the more elementary movement of wiping off the offending substance. A stronger still liquid excites both actions at the same time, and the spinal reflex now interferes with, or inhibits, the higher bulbar mechanism. We may mention here that Eckhard and Steiner's experiments do not support Goltz's statement, that heating the water in which a decapitated frog is placed does not call forth any movements, as it does in the case of an animal deprived of its encephalic ganglia only.

The behaviour of normal and prepared frogs when placed in water offers several points of interest, and is discussed in a separate chapter by Dr. Steiner. He shows that it is neither skin excitations due to the contact of the liquid, nor impressions from the deeper structures due to the loss of weight, which alone start the swimming reflex; it may be both, but the water must also be in motion. The quantity of air contained in the lungs is an important factor in aquatic locomotion, and experiments tend to show that a special respiratory mechanism exists in the base of the optic lobes.

Observations as numerous as conflicting are recorded in the literature of the subject concerning the forced movements following asymmetrical lesions on the frog's brain. Dr. Steiner has gone over the whole ground systematically, and his results are particularly worthy of attention. These forced movements are of three kinds: circular, revolving, watch-hand-like. The first ("manège" movements, gyrations) consist in progression (walking, jumping, or swimming) in a circle; the second in rotation, in a corkscrew-like manner, upon the axis of the body; the third in rotatory lateral movements of the body about its caudal end as a pivot.

Every one is agreed upon the negative results of the ablation of one hemisphere; but when we come to the *thalamus* we find some of the older authorities (Flourens, Renzi) speak of circular movements towards the side of the lesion, whereas Steiner gives as the result of very numerous and very accurate removals of that organ, the supervention of watch-hand rotation towards the opposite side. The probable solution is that, in the former case, the lesions (which lacked exact post-mortem confirmation) extended somewhat beyond the thalamus into the base of the lobe. After a time this rotation ceases, the frog remains quiet, and on excitation, performs circular excursions, also towards the sound side. Within twenty-four hours, locomotion becomes rectilinear again, and the animal is apparently in a normal condition.

Ablation of the roof of one *optic lobe* gives no result; but that of its base gives rise to well-marked gyrations towards the normal side, whether the animal be on land or in water. After a time exhaustion sets in, and the head only remains for a time depressed and turned in the same direction. These phenomena are permanent, and may be brought out for an indefinite time, all the movements being perfectly coördinated. It must be noted that if the operation is not perfectly carried out, and a small portion of the base is left *in situ*, there may be feeble circular and revolving movements towards the injured side.

Removal of half the *cerebellum*, and hemisection of the *medulla* far back near the calamus scriptorius, do not produce any motor disturbance. If, however, the anterior region of the latter organ is submitted to the same operation, muscular spasms are produced, and the frog, on being laid on the ground, jumps repeatedly perpendicularly upwards, and falls on the wounded side. Next comes a period of forced position of the head, which is rotated downwards towards the same side. The opposite fore-limb is rigidly extended to the ground. Placed in water, the frog first performs gyrations away from the lesion, then rolling movements towards it, or the rolling begins at once. All these phenomena are repeated whenever the animal is interfered with, for weeks after the operation. Further researches led Dr. Steiner to discover the exact lesion required to obtain continual rolling, as opposed to the periodical variety just described, in which there is an alternation of circular movements. It consists in a section across the thick edge of the bulb, enclosing the 4th ventricle, at the level of the trigeminus root, a little below the inferior edge of the cerebellum. It may be noticed, by the way, that no paralysis follows bulbar hemisection in the frog.

These results demonstrate that forced movements occur only after lesions of the thalamus, or lobe, or one side of the bulb; and that whilst circular and revolving motions are direct effects of the destructive lesion, watch-hand rotation belongs to the category of the indirect effects due to the traumatic irritation of one lobe. Destruction of the visual centre (roof of the lobe) produces no motor disturbance, though in the frog the optic decussation appears to be complete. The perfect coördination in swimming shows that forced movements are unconnected with paralyses or contractures; and the fact that these movements do not arise without some external excitation, proves that the term "forced" is not to be taken in Magendie and Vulpian's sense, who assumed the existence of a kind of internal impulse within the nerve-centres. It would be more consonant with the facts to interpret these phenomena as muscular adaptations, the response to centripetal misinformation, so to speak, through disturbances in the receptive and elaborative centres.

Dr. Steiner describes the results obtained by hemisections at different levels, and in different directions of one thalamus, or optic lobe, for the details of which we must refer the reader to the original. One point is of interest—viz. that longitudinal division of one lobe is immediately followed by "manège" towards the opposite side; but within twenty-four hours the frog appears to be normal again in every respect, whereas a diagonal section produces permanent effects. These facts agree with the view taken by the author of the functions of the basal part of the optic lobes as end-stations of centripetal tracts. It will have been noticed that rolling movements always take place to the wounded side, even when combined with gyration in the opposite direction. Dr. Steiner concludes this portion of his work by showing that no forced movements follow bulbar lesions after removal of the commissural part, which he once more declares to be the only encephalic motor centre.

The experiments of Professor Fano on the brain of the tortoise (*Emys europæa*), were made contemporaneously with those of Dr. Steiner; and the results of these two observers gain in value and interest from having been made independently and under different conditions. The most striking fact connected with the cerebral mechanism of the tortoise is that when the animal is deprived of the whole encephalon, except the medulla oblongata, it performs for a considerable period, either at intervals or continuously, coördinated locomotor movements, without intervention of any external stimulus. These movements persist, though the animal may be arrested by an obstacle, and are of two types: either the opposite front and hind-legs strike out together, as in normal locomotion, or the rhythm is altered, and the hind-legs simply push the body forward, making it pivot on the shoulder-joints of the fore-limbs which have been previously placed, fully extended, upon the ground. These two gaits occasionally alternate in the same subject; but we are not informed of the anatomical or other causes to which this variation may be due. The movements of the brainless tortoise are throughout characterised by an expenditure of energy disproportionate with the effects produced; whilst moving, it holds its head and neck extended, and performs lateral movements of the tail.

The direction followed by such animals was studied by making them mark their paths on the floor. It is thus shown that they never walk in a straight line, but describe either undulatory curves, or irregular circles to one side, as if there was always one half of the nerve-centres acting more than the other After being shaken, the prepared tortoise always describes an "initial loop," or more or less complete circle, before resuming its ordinary mode of progression. Professor Fano also shows that in those animals which display the phenomenon of intermittent deambulation, there is no definite relation between the periods of rest and the activity during those of walking; and compares in this respect the action of the bulbar locomotor centres to that of the respiratory and cardiac centres in certain abnormal conditions.

The ordinary reflexes are more easily obtained in the hind-legs, whereas the coördinated locomotor movements are more marked and persistent in the fore-limbs. In one case of accidental injury the opposite condition was found to obtain; and here the action of the posterior extremities was altered, and of a retrogressive nature. Professor Fano was not able to obtain the same results experimentally, which reminds us of what happens in the frog after removing the anterior two-thirds of the basis of the optic lobe.

The motor mechanism of the medulla oblongata resides in the anterior two-thirds of the organ, for when these are separated from the cord, every movement ceases. A longitudinal median incision does not interfere with this activity. Decapitation is followed in the tortoise (as quite recent experiments have shown it to be the case in the duck also) with a few short-lived locomotor movements, as if the motor spinal elements had preserved an impression, as it were, of the higher coördinated impulses.

The occurrence of spontaneous movements in the brainless emys shows that we cannot generalise the results obtained on the frog and other animals, and speak of the encephalic centres as essential to their production. It also led Professor Fano to extend his experiments to other cold-blooded vertebrates; and after some failures, he found that the same phenomenon could be observed in toads (*Phryne vulgaris*, but especially *Bufo viridis*). Shortly after removal of all the encephalic ganglia, with exception of the bulb, the toad begins to walk (not to jump) with the ordinary crossed action of the anterior and posterior limbs; the latter, as in the tortoise, manifesting a slight delay. The gait is more flaccid than in the healthy animal, and obstacles do not arrest the movements. This ambulatory activity is never continuous, but always alternates with periods of rest, and persists for many days. The graphic method reveals phenomena essentially similar to those observed in the tortoise as to the paths described by the animals.

Successive transverse sections of the bulb prove that the motor mechanism is localised in its anterior portion, but seems to extend farther back than the corresponding centre in the frog, so that respiration ceases together with locomotion. The proximity of the locomotor and respiratory centres led the author to ascertain whether there is any relation between their activity in the toad as well as in the tortoise; but simultaneous tracings show no regularity in the collateral course of ambulation and respiration.

Professor Fano believes that the activity of the locomotor bulbar mechanisms does not depend upon any peripheral excitation, is not a reflex, but rather an expression of nutritive activity in the centres. We cannot here adduce the various arguments by which he endeavours to support his view; but he seems to underrate the influence of the centripetal sub-conscious impulses from the organism itself as a possible source of excitation. At any rate, several of his remarks are quite reconcilable with the opposite explanation of his facts, whilst they receive no support from the results obtained on other animals.

Removal of the *cerebellum* does not appear to have more effect in the tortoise than we have seen to be the case in the frog; the *optic lobes*, on the other hand, appear to play an important part in the motor manifestations of the former. Their presence, after removal of the hemispheres and thalami, is sufficient to arrest the locomotor activity of the bulb; and systematic sections show this influence to be exerted by the basal portion of these organs. On the other hand, removal of the optic lobes only produces great restlessness in the movements of the animal, which, on being interfered with, no longer withdraws into its carapace, or even slackens its pace. Professor Fano distinctly asserts that tortoises can see so as to avoid obstacles, after complete ablation of the lobes; and accepts Setschenow's theory, that these organs inhibit reflex movements by retarding the transmission of excitations through their ganglionic structure. It is difficult to reconcile these observations with the results of similar experiments on frogs and fishes.

Ablation of the *hemispheres* in the tortoise does not appear to suspend spontaneous movements; the animal walks, swims, and acts as a normal specimen, only more slowly. Electric excitation of these organs, if protracted for a minute or so, is followed by a short period of active walking, during which the animal does not respond to external excitation, "as if the influence of the optic lobes had been suspended." The same phenomenon can be elicited several times in succession. Electric excitation of the thalami in the whole animal produces symptoms of pain, the limbs being violently withdrawn within the carapace. The same excitation, after removal of the hemispheres, and protracted for a sufficient period, may give rise to feeble locomotion, which becomes more pronounced upon ablation of the optic lobes.

Unilateral lesions of the brain in tortoises does not give rise to any such varied and marked forced movements as they do in the frog. Removal of one hemisphere, as usual, is ineffective; but when the corresponding lobe is taken away also, circular or "manège" movements are observed, the animal's walk being in large circles towards the opposite side. There is no hemiplegia, and the posterior extremities move harmoniously; but an alteration is perceptible in the coördination of the two anterior limbs. The lesion being on the right side, the left leg performs movements of adduction, the right of abduction, after having been placed on the ground, and the circular movements are to the left. Professor Fano does not account for these phenomena on the theory of forced movements, but assumes that there are in each hemisphere and lobe, centres for the adduction of the leg on the same side, for abduction of that on the other. He finds that on dividing the cervical cord longitudinally along the middle line, the animal walks in larger circles, and that there is less abduction of the right leg; and he explains the latter fact by assuming a partial decussation of the motor fibres in the cord. On continuing the incision upwards, so as to bisect the medulla in its whole length, the animal starts upon its gyrations in the opposite direction; the left fore-leg continues its movements, whilst the right hind-leg is motionless, and the right fore-leg performs large ineffectual excursions, which cease on removal of the motor bulbar centre. We do not see any difficulty in explaining these phenomena on the theory of forced movements; and, without assuming the unlikely existence of adductive and abductive motor centres in the fore-brain, in accounting for the facts by an abnormal action of the bulbar centre dependent upon a disturbance in the higher receptive and regulative centres.

The general result of the most recent researches on the cerebral functions in amphibians and reptiles is thus that the greater part, if not the whole, of the encephalic motor centres is contained in the anterior part of the medulla oblongata. The hemispheres apparently contain the elaborative mechanisms forming the physical substratum of the higher conscious processes. The thalami seem to be connected with cutaneous, the basis of the optic lobes with motor, feelings. The influence of the cerebellum on coördination is but slight in the lower classes of vertebrata; the researches of Baudelot[1] on fishes had already demonstrated this fact, which the small development of that organ might have led one to anticipate. The importance of these results towards the elucidation of many problems of cerebral localisation in man can hardly be overrated; and we may look to the comparative method of investigation for much light and assistance in the future.

A. DE WATTEVILLE.

MEYNERT (THEODOR). *Psychiatrie. Klinik der Erkrankungen des Vorderhirns begründet auf dessen Bau, Leistungen, und Ernährung.* Erste Hälfte mit 64 Holzschnitten und 1 Tafel. Wien: Wilhelm Braumüller, 1884.

MEYNERT (THEODOR). *Psychiatry, a Clinical Treatise on Diseases of the Fore-Brain, based upon a study of its Structure, Functions, and Nutrition.* Translated by B. SACHS, M.D. Part I. New York and London: G. P. Putnam's Sons, 1885.

A WORK from the pen of Prof. Meynert on the Diseases of the Cerebrum is an event of no small importance, and the English reader is under a deep debt of gratitude to Dr. Sachs for placing within his reach a translation of it, which is as accurate, clear, and readable as any translation of Meynert's writings is ever likely to be. Those who are acquainted with the obscure and involved style of the original will readily understand that Dr. Sachs' task has not been by any means an easy one, and will learn without much surprise that "the attempt at a literal translation had to be abandoned." The first half of the first part of the work—the only part which has yet appeared—deals with the structure, architecture, and minute anatomy of the brain. For nearly a quarter of a century Prof. Meynert has been instructing us in the anatomy of the brain, and much of our present knowledge of the structure of the organ is due to his labours. But much has happened during this time, and it is possible that the teaching which was new and in advance of the age in the beginning, is old-fashioned, and behind the age at the end of the period. The method of investigation adopted by Meynert—the old cleavage method—is by no means a perfect one, and it is only in the hands of a thoroughly practised expert like himself that anything like trustworthy results could be obtained by it. And although we believe the statements of Meynert with regard to the structure of the brain to be on the whole accurate, yet we have no hesitation in rejecting any of his conclusions which are found to be in conflict, and not a few of them are in conflict, with knowledge obtained by means of better methods than the one he adopts, such as the method of embryology, as practised by Flechsig; that of arrest of development, as introduced by Gudden; and that of secondary degenerations, as employed by Türck, and Charcot and his pupils.

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[1] See 'BRAIN,' April 1884, p. 133.

But even after making many deductions for the shortcomings of this work, the reputation of Meynert as a great anatomist is, on the whole, thoroughly sustained in it. There are not many anatomists who would find much scope for the exercise of their talents in giving a description of the basal surface of the brain; but nowhere probably is the particular genius of Meynert seen to greater advantage than in furnishing such a description. The small print on page 26 (page 28, transl.) we regard as a peculiarly favourable specimen of his work. The author's unrivalled knowledge of the comparative anatomy of the brain has enabled him to compress in a few lines a truly astonishing amount of information. Every sentence is a generalisation, and every generalisation helps to explain the structural and functional differences existing between the human brain and the brains of the lower animals. Many pregnant passages of this kind are to be found in the work before us, and in some few places new lines of investigation appear to be opened up, which will be sure to bring a great reputation to any one who may have the sagacity to see their significance and the industry to pursue them.

The next section of the work is entitled "Anatomical Corollaries and Physiology of Cerebral Architecture;" but it really treats of the relations existing between the structure of the brain and mental processes. In order to discuss this subject profitably an author ought to be not simply a great anatomist, but a great subjective psychologist also, his knowledge of psychology being attained either by the analysis of his own thoughts and feelings, or by the analytical labour of others. Judging from the work before us, we have no hesitation in saying that Prof. Meynert's knowledge of subjective psychology is an exceedingly slender one, and we pass upon him a very temperate judgment in saying that his psychology is fully a generation behind the psychology of this country as represented by the writings of such men as Bain, Spencer, and Hughlings-Jackson. But temperate as we believe this judgment to be, it will be expected that some considerations in its justification will be advanced. Let us take as a test the principle, or rather postulate, with which the author starts what he calls his anatomical corollaries. He asserts that "the only specific energy of brain cells is a faculty of sensation (Empfindungsfäigkeit, translated by Dr. Sachs 'sensitiveness'), which is converted in the forebrain into actual sensation." But to postulate the existence of sensitiveness or of a faculty of sensation, in order to account for the fact of sensation, appears to us to be an attempt to explain the *obscurum per obscurius*, and when Meynert proceeds to apply and illustrate this principle, the result appears to reach the superlative degree of the obscure. Surely no one is assisted in comprehending the connection existing between the activity of brain cells and sensation by being told that "in assimilating totally unknown physical impulses, the cerebral cortex and complicated protoplasmic structure, resembles the protoplasm of the primitive amœba, which can transform itself into a hollow mass, and can thus encircle any body which it wishes to assimilate." This comparison may be regarded as a good metaphor, but has no claim to be regarded as a scientific generalisation.

The next principle which Meynert advances is "that the processes of innervation from the hemispheres, which constitute what we term *volitional* acts, are nothing more than the perception, or memory of sensations of innervation (Innervationsgefühle.)" This doctrine is not very intelligible as it stands, but the context shows that by "processes of innervation from the hemispheres," Meynert means the motor actions which are set up by excitation of certain areas of the cortex of the brain, and by "sensations of innervation" he means the sensations which accompany muscular effort, or, in other words, the muscular sense. This principle, therefore, in reality asserts that the so-called motor centres of the cortex are not motor centres at all, but are sensory centres of the muscular sense. This is the theory which has been adopted by Munk, and which has also played a considerable part in the explanations of Goltz and other German physiologists. A good deal of ingenuity is manifested by Meynert in illustrating this theory, but it must still be regarded as a plausible hypothesis which has not yet received its verification in facts; and even if all the author's statements were acknowledged to be true, the only conclusion warranted by them is that the cortical sensory centres for the muscular sense coincide with the centres from which the centrifugal conducting paths for volitional movements take their origin. Some few pathological facts can be adduced in favour of this opinion, whilst others might be adduced which are not in accord with it. None of these, however, are mentioned in this part of the work, but we may expect to find them discussed in the clinical part. But the known facts that centrifugal conducting paths emerge from certain areas of the cortex, as is indeed shown on Meynert's own diagrams; that the fibres of these conducting paths spring from large caudate cells more or less similar to the motor cells of the spinal cord; that the areas in which these cells exist give a motor reaction to electrical excitation, while the areas in which they are absent give no such response; and that destruction of the areas with the large pyramidal cells gives rise to a descending degeneration of the conducting paths, which can be traced into the spinal cord, while destruction of other areas is not followed by any such result, combine to prove in the most conclusive manner the propriety of designating areas which are endowed with such properties by the name of motor centres; and we believe that every argument which has been adduced by Meynert in opposition to this conclusion is merely so much misplaced ingenuity. But if any one is enamoured of Meynert's theory of the motor centres, he had better see how he likes it when applied to the explanation of the phenomena of disease. The author mentions the case of a boy who was affected with a large tuberculous tumour of the mesencephalon and thalamus. In addition to paralysis of the right third nerve, and of the left facial nerve and left extremities, it was noticed "that the head of this boy was turned towards the left side, and that, while at rest, there was flexion of the left upper extremity, and extension of the right upper extremity." The following is the author's explanation of this forced attitude:—"We may suppose that the boy," says Meynert, "lacking the innervation-sensations of the left rotatory muscles of the spinal column, was induced to excite the missing sensation of innervation by a volitional rotation, to produce the lacking sensation of innervation of the left arm flexors by forcing a flexion of this arm, and under similar circumstances to compensate for the disturbed sensation of innervation of the right extensor muscles by exciting a forcible extension of the right arm. The lack of innervation-sensations created delusions with regard to the position of his extremities, which in turn gave rise to volitional movements, in consequence of which he assumed the typical pathological position, the so-called forced position." The above passage is quoted from Dr. Sachs' work (p. 165), which appears to us, on comparison with the original, to be a very faithful and literal translation of the author's words; but we have to confess our complete failure to discover from a study, either of the original or the translation, any meaning whatever in this pretended explanation. To us it seems the most pronounced example we have met with for a long time of high-sounding jargon, but others may be fortunate enough to find in the passage the wisdom which is hidden from our view.

After passing such a severe judgment—a judgment which we shall be glad to see reversed by a more fortunate or more discriminating critic than ourselves—it is a sincere pleasure to acknowledge that the author's remarks on the functions of the association system of fibres are both judicious and well worthy of study. His theory of the functions of these fibres is practically the same as that enunciated by Mr. Herbert Spencer many years ago. That theory is that the activities of the cells of the brain are the correlatives of the feelings, and that the activities of the fibres are the correlatives of the relations between the feelings. But although the theory has been stated in general terms by Mr. Herbert Spencer, his practical acquaintance with the anatomy of the brain was too scanty to enable him to connect the relation between the feelings with a definite system of fibres. This was done by Meynert as far back as 1865, when he showed that the association fibres are connected at both ends with cortical cells.

Meynert agrees with Munk in believing that every portion of the cortex of the brain subserves intellectual functions, and he condemns the opinion of Hitzig, who placed the intellectual functions in the frontal lobes. It never seems to occur to him that it is possible for both of these opinions to be true, according to the extension given to the term "intellectual functions." As we sit writing, our eyes are raised, and we judge that a book lying on the table is three feet distant—Newton judged that the earth was ninety-one millions of miles distant from the sun. Both judgments are reached by the exercise of intellectual functions, but are the judgments themselves on the same level of intellectuality? Surely not. Our judgment was the result of direct perception, and was made in all but an unconscious manner; while Newton's was the final result of an involved and intricate train of indirect reasoning, every step of which required that highest degree of consciousness, called close attention. Now if the direct judgments, which are involved in every act of perception, are to be regarded as being reached by the exercise of intellectual functions, and this is certainly the most scientific conception of the intellect, then it is highly probable that the whole of the cortex ministers to intellectual functions; but if those judgments which are reached by indirect reasoning are alone entitled to be regarded as resulting from the exercise of intellectual functions, and this undoubtedly corresponds to the popular conception of intellectual processes, then the intellectual functions, and most probably the higher emotions are, in our opinion, localised chiefly in the anterior lobes of the brain. In one word, the difference between Meynert and Munk, on the one hand, and Hitzig on the other, is one of terminology, and not of fact. Meynert is equally unfortunate in his references to the localisation of memory. "No author of the present day," he says, "would be likely to insist on one special seat of memory, for memory is the common property of all cortical cells and fibres which are able to receive and conduct external stimuli of all sorts." This assertion is, in our opinion, true; but it is such a one-sided presentation of the facts, that it can hardly be distinguished from an absolute error. We do not believe, any more than Meynert, that memory is localised in any circumscribed area of the brain; but we do believe that particular forms or manifestations of memory are so localised. Let us take the famous American crow-bar case, in which the frontal lobes were almost completely destroyed, as an example. The man's memory for events and dates was not sensibly impaired after his recovery, but his moral character having undergone marked deterioration, it is very likely that such words as "baseness," "self-denial," "justice," and "righteousness," would not after the injury call up in his mind the same intricate associations as they did before it. It may therefore be said that the patient suffered from an impairment of memory, although to ordinary observers loss of memory was not a prominent symptom. Let us contrast the symptoms in the case just related with those of a case in which there is a spot of softening in the left hemisphere of the brain in the convolutions which border the horizontal limb of the sylvian fissure—the first temporo-sphenoidal and the supra-marginal convolutions. Under such circumstances patients suffer from word-deafness, or, in other words, spoken language ceases to have any meaning for them, and most, if not all, of these patients are the subjects of what Kussmaul calls the aphasia of recollection, a condition in which the patient is unable to name his own bodily organs, or any single object presented to him. In the face of facts like these, how futile it is to assert, without any qualification, that memory is the common property of all cortical cells and fibres!

In his Preface the author tells us that he has deemed it necessary to criticise "Darwin's theory of the inheritance of acquired faculties." The promised criticism of this doctrine is in the following terms:—"The Darwinian doctrine that ideas are inherited, and are not the result of perception and association; that movements, even mimical ones, are the result of innate motives, and have nothing to do with imitation and early reflexes, can hardly be applied to man. Not even his upright gait, which is surely a universal form of movement, is innate in man; it is acquired with difficulty only through imitation and cortical coordination."[1] So far as can be gathered from the present work, Meynert is an adherent of the empirical school of philosophy, and he would have us start our explanations of mental phenomena with the *tabula rasa* of Locke. But if he denies the existence of notions native to the mind, or innate ideas, then he has to combat not Darwin's views, but the whole of Kant's transcendental æsthetic, and the transcendental logic, or, at any rate, the first part of it which constitutes the transcendental analytic. He will also have opposed to him every philosopher of note since the time of Kant, with the exception of Brown, Bentham, and the Mills in this country, and possibly Comte in France, but with, so far as I know, no exception whatever in Germany. It is not, therefore, to be expected that any author, however gifted he may be, can dispose of the theory of *à priori* notions, or innate ideas, in a couple of sentences. But if a critical analysis of our thoughts and feelings shows that some, not all, of our ideas are *à priori* or innate, the question then arises—have these ideas been implanted in our minds by a special creative act, or have they been formed by a progressive development? The existence of innate ideas was used very effectively a few years ago in this country by the opponents of the Darwinian theory, and in all their arguments they assumed that these ideas were a specially created endowment of the human mind. And it was not Mr. Darwin, but Mr. Herbert Spencer, who advanced a theory of innate ideas, which brought them into conformity with the general doctrine of evolution. Mr. Spencer did not deny the existence of innate ideas, but he showed that, although these ideas could not have been generated by the experiences of any single individual, yet they were produced in us by the accumulated and inherited experiences of the race This doctrine has reconciled the differences between the empirical school of philosophy, which took its origin with Locke, and the intuitive school which dates from Kant, and it has produced a profound revolution in psychology, as well as in philosophy—the most profound and far reaching, indeed, which this century has witnessed in any department of knowledge; and if Meynert chooses to attack this doctrine at any future time, we hope he will grapple with it as it is presented by its original author. No one denies that individual experience is necessary in order to develop even innate ideas, just as no one denies that light is necessary to develop sight, although our ability to see depends chiefly upon the fact that we are born with eyes. And similarly, although a child requires a considerable amount of training before he can maintain the upright posture, the fact that he can maintain it with ease, after such training, results less from the training than from the structure of his body; and this, in its turn, depends upon the innate tendency the organism has to grow into the parental shape.

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[1] Page 256, trans. p. 274.

Nothing could show more convincingly the utter incapacity of Meynert to deal satisfactorily with subtle psychological problems and philosophical discussions, than the manner in which he jumbles in inextricable confusion such words as "the ego," "the individuality," and "the character." The ego, according to Descartes, is the thinking substance in man, in contrast with the extended substance of which his body and the surrounding world are composed, and the word has been used in this sense by almost all subsequent philosophers. Most philosophers would refuse the possession of an ego to the lower animals, and we know of no author that would accede the possession of it to vegetables. Individuality, however, belongs to vegetables as much as to man, and indeed, some biologists believe that the individuality or the principle of individuation is coextensive with living beings, and they have accordingly included the principle in their definition of life. In its primary meaning the word "character" refers to the moral qualities of a man, and in its secondary meanings, a good or bad character is given to animals, vegetables, and even to inanimate objects according as they are useful or injurious to man. It will thus be seen that "the ego" is a philosophical term to indicate the mind in opposition to matter, that "the individuality" is a biological term which refers to all living beings; and that "the character" in its primary meaning, is a term of moral science, although in its secondary meanings it has a more extended application.

Let us now listen to what Meynert has to say with regard to these terms. He believes that there is a primary ego which is defined by the limits of the body, but he continues, "the primary ego expands through permanent and intense secondary perceptions joined to it by association; so that intimately related persons, property, skill obtained by constant practice in art, science, a fondly cherished aim in life, convictions, patriotism, and honour become part of the ego" (p. 175, trans.). When speaking of the cortical centres, he says—"the sum of these 'centres' constitutes 'the individuality,' the 'ego' of abstract-psychologists;" and a little further on he adds, "this unequal activity of the fore-brain, constituting individuality, varies as regards contents and degree with each person; it is designated also the *character* of the individual" (p. 167 et seq., trans.). Our wealth of philosophical language is not so great that we can, without offering the strongest protest in our power, allow words which have had definite meanings assigned to them for generations to be sacrificed in this rude manner by having their differences effaced, and their connotations reduced to an identity.

In the next section of the work Meynert treats of the "Nutrition of the Brain," and his remarks on this subject will be found to be less obscure and much more worthy of consideration than anything we have met with in his psychological discussion. In his theory of disordered nutrition, however, he attaches much too great importance to the action of the vascular system with its nervous mechanisms, and too little to the action of the nervous tissues which are the seat of the abnormal changes.

The first part of the work is brought to a close by an Appendix on the "Mechanism of Expression," and of it we shall only say that the readers of Darwin, Spencer, and Bain will gain nothing, but will lose much by adopting Meynert's interpretations. An author who rejects altogether the theory of "the inheritance of acquired faculties" cannot be expected to throw much light on the mechanism of expression, and accordingly Meynert does not afford us much enlightenment; but on the contrary, he casts the haze of his heavy and obscure style over this subject, which is all the less excusable inasmuch as it is treated with such pellucid clearness in the charming pages of Darwin.

James Ross.

Illustrations of Unconscious Memory in Disease, including a Theory of Alteratives.—By CHARLES CREIGHTON, M.D. 1 vol. 8vo. pp. 212. London: Lewis, 1886.

THIS work of Dr. Creighton constitutes an important addition to our stock of pathological knowledge, it assists us in the interpretation of many morbid processes, and will no doubt be further developed in the hands of the author. The term "unconscious memory" is one not altogether unfamiliar to us; modern research into such subjects as instinct in animals, the automaton action of infants as well as several acquired habits of adults, have led us to look upon many complex movements as the result of mechanisms without any aid of thought or consciousness. If such an act as playing the piano may occur whilst the mind is wandering, and the performer scarcely conscious of it, we cannot avoid the conclusion that the memory is still awake, and therefore the appropriateness of such an expression as unconscious memory. The somnambulist may perform some wonted act and not be conscious of it; in this case the memory cannot be said to be lost. In the same way a morbidly acquired habit may persist long after all consciousness of the first voluntary act has passed. In the case of a young animal choosing its food, it may be said that the memory of a like act was transmitted by its predecessors, and there are some strong upholders of Darwinian doctrine who would account in this way for all inherited instincts. Instead of saying that instinct guided it, a more intelligible expression would be that the animal retained a memory of the past.

For some years we have employed the expression in the instruction of students, having been more especially indebted for the use of it to the author of "Life and Habit," a gentleman, it will be remembered, who contended for the priority of some of the ideas enunciated by Darwin. He would not say, for example, that instinct compelled the chick to break its egg at a certain period of its development, but that the occupier of the shell remembered the particular day and manner in which its parents broke through their covering, and made their escape. We have thus found the term very useful in the explanation of the continuance of many diseases, especially those of the nervous system. An acquired habit in an individual is nothing more than the education of a spinal centre for a particular act or to a new function, and in this sense habit is second nature. A choreal movement which lasts during life may be regarded as a newly acquired function of some part of the nervous system. An analogous condition of the sensory centre would afford an explanation of a pain which was altogether subjective. If motor cells, instead of being quiescent, had been taught to act rhythmically and regularly like those of the respiratory centre, a new and peculiar movement would result; and, in a similar manner, if some central sensory cells remained in a polar condition after the natural distal stimulus which excited the action was removed, the pain would nevertheless continue, and be styled in ordinary language subjective.

Having already had our mind directed towards some of the propositions and theories propounded by Dr. Creighton, we were the better prepared to consider them than if they had dropped upon barren ground as a pure subject of speculation, and thus we can with the more confidence give our support to this most valuable contribution towards an explanation of many of the abnormalities of the human body. The doctrine is a distinct offshoot of the evolutionary and Darwinian theory, showing how fruitful has been the new departure of thought in every department of science.

The author has adopted the term from Hartmann's "Philosophy of the Unconscious," and from Hering's work on "Memory as a General Function of Organized Matter." Dr. Creighton's object is to show how a large number of pathological states are explicable on the idea of the continuation of the memory of past conditions or actions, whether these be in nerve centres or in secreting organs. He gives a large number of examples where a morbid state is perpetuated when once started into action, and even when the original cause is removed, and out of this highly scientific and philosophic idea, speculative as some may think it, a highly practical result ensues, and in this respect Dr. Creighton's book may rival many works on pure therapeutics; the theory of cure being simply this, that a tissue has taken on a morbid or unusual habit, and the medicine breaks the habit. Herein lies the explanation why so many different remedies may be found for curing or averting disease. We might take, for example, the cure of phthisis by various remedies, such as altered diet, a sea voyage, or residence on an alpine height. None of these means could act otherwise than breaking up the tendency to morbid action in the lungs by altering all the circumstances which first promoted it. If a young man shut up in an office and surrounded by various unfavourable conditions falls into ill-health, a change of his environments would arrest the morbid process which had begun. This may be true of many other complaints as well as of phthisis; the remedy, change, being especially enforced in this case, because it had been thought that the disease being one of the lungs, the most essential element of cure was to be found in good air. Various diseases are thus cured by breaking up the habit which was fast tending to develop them. The author's theory recalls to mind D'Alembert's notion of the operation of medicine; that nature is fighting with a club, when the doctor steps in and fights with his club; the kill or cure depends upon who has the mastery. Dr. Creighton, in like manner, thinks that the so-called alterative medicines set up such a commotion in a part of the body which is not acting normally, that after the disturbance is over, it may then go on right again and pursue its legitimate course. In some cases the medicine may produce a corresponding action with the existent morbid one, and in this way drive it out according to the homœopathic doctrine; in other cases the medicine may set up an opposite action, and get rid of the old process by mere antagonism; in still other cases the kind of disturbance produced by the drug is not clear, but the result is that good eventually comes out of it. It is sufficient to break the habit which has made some part of the animal machinery work in an abnormal manner.

The therapeutist, therefore, as well as the pathologist, will hail Dr. Creighton's work as affording him fresh encouragement for the use of drugs. For, as the author mentions, the cause of a malady may be known, and be actually removed, and yet the old habit remain until some powerful drug come into play to rouse the morbidly-acting tissue, and make it shake off the newly-acquired process. The strongest argument in favour of drugs may be found in the fact, that nothing but some very powerful agent can arrest many diseases, or, as they may be called, morbid habits.

Much more is to be found in Dr. Creighton's work than our time or space will allow us to allude to, therefore it should be read not only by those who are anxious to add some fresh knowledge to a special subject, but all who are interested in the scientific advance of medicine as it advocates fundamental truths, which lie at the very foundation of our science and art.

As before remarked, it is an offshoot from the modern mode of thought which has tended to promote scientific advance in every direction. Of this Englishmen may be well proud, for although in a large number of departments of science England is overwhelmed by the crowd of continental workers, yet our country has always stood foremost in the production of original thinkers and of men who have given a new point of departure in modern discovery. The last twenty or thirty years has witnessed a complete revolution in the mode not only of considering inorganic nature, but of the organic world also, including man and all his attributes. For this, civilization is chiefly indebted to such men as Darwin, Herbert Spencer, and Lyell.

Samuel Wilks.

Klinische Psychiatrie; specielle Pathologie und Therapie der Geisteskrankheiten. By Dr. Heinrich Schüle, of Illenau. 3rd ed., 534 pp. Leipzig: F. & W. Vogel, 1886.

The two former editions of this book appeared as a volume of Ziemssen's 'Encyclopædia.' This is an almost entirely new book, the author's object being to give an exhaustive description of the clinical symptoms of insanity, including their causes, prognosis, and treatment; whereas the subject of pathological anatomy is only dealt with in the chapters on general paralysis and idiotism.

Dr. Schüle's classification of mental diseases is founded upon a new principle; he maintains that we have to distinguish them according to the condition of the brain at the time of the onset, inasmuch as the resistance of the brain depends upon whether the organ is in a "healthy" (*rustig*) or an "invalid" condition. From this point of view the author establishes the following system:

I. MENTAL DISEASE OF THE DEVELOPED BRAIN.

1. *Diseases of the "Healthy" Brain (Psychoneuroses):*
   (*a.*) Melancholia.
   (*b.*) Mania (mitis and typica), with secondary insanity (dementia).
2. *Diseases of the "Invalid" Brain (Psychoneuroses):*
   (*a.*) Mania gravis, Furor.
   (*b.*) Paranoia (acute, chronic, atonic—Kahlbaum's Katatonia.)
   (*c.*) Acute primary dementia.—Appendix: Hallucinatory stupor.
   (*d.*) Hysteric, epileptic, hypochondriac insanity.

Appendix: (*α*) Periodic, circular, alternating insanity; (*β*) insanity caused by extra-cerebral disease (puerperal insanity, &c.), and intoxication.

3. *Pernicious Exhaustion of the Brain:*
   (*a.*) Acute exhaustion—delirium acutum.
   (*b.*) Chronic exhaustion—dementia paralytica.
4. Insanity produced by organic brain-disease: modifications of progressive paralysis.

II. MENTAL DISEASE ON THE BASIS OF MORBID DISPOSITION, OR DEFECTIVE CONSTITUTION.

(*a.*) Hereditary neuroses—Appendix: Transitory insanity.
(*b.*) Simple form of hereditary insanity—Maladie du doute et du toucher, &c.
(*c.*) Primary paranoia—Delusions.
(*d.*) Moral insanity (degenerative hereditary insanity).
(*e.*) Idiocy.

It hardly needs to be said that this system, like all other systems, has its deficiencies; so we find "mania" among the diseases of the healthy brain as well as among those of the invalid brain; the type of primary (originary) paranoia is severed from the other forms of the same clinical character; nevertheless Schüle's classification opens many new points of view which are of great value for the estimation of the different forms of insanity, especially for their prognosis. The greatest difficulty will always be to find out in a given case whether one has to deal with a "healthy" or an "invalid" brain; the determination of this point must often be a matter of mere individual impression.

Dr. Schüle's descriptions of clinical cases are most instructive, full of life, the result of long and careful observation; the author's desire to avoid the conventional phraseology of text-books gives his style a peculiar stamp, but it must be recognised that this new edition of the book is very much more readable than it was in its former shape.

It is impossible to give here a detailed account of the contents of the book; may it suffice to say that in our opinion Schüle's 'Klinische Psychiatrie' is the most valuable work on the subject which has been published in Germany since Griesinger's classical work: 'Die Pathologie und Therapie der psychischen Krankheiten;' and that we strongly recommend it to the attention of our readers. R. H. PIERSON (Pirna, Saxony).

Allgemeine Diagnostik der Nervenkrankheiten.—By Dr. P. J. MOEBIUS. 1 vol. 8vo., 338 pp. Leipzig: F. & W. Vogel, 1886.

THIS book is intended to serve as an introduction to the study of neuropathology, and it will be found very useful for the purpose. The standard works on the diseases of the nervous system require the knowledge of the different methods of examining a patient with regard to the condition of his nerves, but they do not teach the student their application in a sufficiently exhaustive manner. Therefore, Dr. Moebius's treatise, which is a plain, easily intelligible and, at the same time, thorough exposition of the subject, cannot fail to meet with much approval.

After a more general instruction concerning the way of examining a patient, taking the family history, &c., the author describes the methods of testing the state of the mental faculties, the condition of speech, chiefly after Kussmaul and Lichtheim, including the disturbances of reading and writing. Then follows the examination of the motor apparatus: active mobility, reflex, mechanic and electric excitability (the latter part after Erb's Electrotherapie). The next chapter is devoted to the examination of the state of the special senses: sight, hearing, smell, taste, and a more detailed account of the different ways employed for stating the condition of sensibility. We quite agree with the author when he finally says that for this purpose the simplest method is the best, and that all those complicated tests which are indispensable in physiological research are more or less unavailing at the bedside.

In an Appendix, the physiology and pathology of muscles, and the disturbances produced by lesions of the different nerves, are treated with much conciseness and lucidity.

Numerous excellent diagrams, partly original, but to the greater part taken from the works of Erb, Duchenne, Charcot, Strümpell, and others, add considerably to the value of Dr. Moebius's book, which is not to be considered as a mere compilation, but shows throughout the hand of a critical and experienced observer.

We gladly take this opportunity to mention, that with the present year Dr. Moebius has assumed the joint editorship of *Schmidt's Jahrbücher*, the 209th volume of which is in course of publication. His name is a token, that neuropathology will henceforth be worthily represented in the familiar pages of this old-established record of medical progress.

R. H. PIERSON.

Abstracts of British and Foreign Journals.

S. Kostjurin on the Senile Changes of the Cerebral Cortex (*Wiener Medic. Jahrbücher*, 1886, Hft. 2).—The author examined a number of brains of old people in Professor Obersteiner's laboratory, in order to establish the histological changes to which the various tissue-elements of both simple and senile atrophy may be referred.

He arrived at the following conclusions :—

1. The great part of the nerve cells in old age belong to a class of a more or less well-marked pigmented fatty degeneration, and show sometimes the formation of vacuoles.

2. The medullated nerve fibres of the cerebral cortex in part atrophy and become fewer in number.

3. The small blood-vessels are affected by atheromatous degeneration, viz. an almost universal spindle-shaped thickening of the connective tissue of their walls, which may result in the entire closing of their lumen. The quantity of the normal amount of pigment present in the adventitia of the arteries seems slightly increased.

4. In place of the atrophied cells and nerve fibres, there is a slight thickening of the connective tissue.

5. At the periphery of the convolutions of the brain there are always a larger or smaller quantity of corpora amylacea, which may form a continuous enveloping layer round the cerebral cortex.

These several degenerative changes are, as regards their intensity, not so dependent on the age of the individual, but rather correspond to the decrease of weight of the brain; in heavy, though at the same time very old brains, they may be nearly absent, whilst in much younger but particularly light brains, they are very marked. This is mostly the case when there are degeneration and decrease of the nerve fibres.

Further, the above described changes of the different elements forming the cerebral cortex will suffice to explain all those phenomena which characterise senility, as far as the activity of the brain is concerned.

One must regard them as the normal result of the brain of old age, and when other pathological appearances are present in such brains they must be referred to other causes than old age.

L. Merk on the Arrangement of Karyskinetic Figures in the Central Nervous System and Retina of Embryos (*Sitzungsberichte der R. Acad. d. Wissensch. in Wien*, 92 B., 1885).—In embryos of the Tropidonotus natrix one finds in the earlier stages the surface of the neural tube, which is turned towards the central canal of the spinal cord, as well as the brain ventricles, studded with karyskinetic figures, whilst in the remaining wall of the central nervous system there are at that period hardly any appearances of karyskinesis to be seen. An important circumstance is that the two new nuclei almost always lie close together, not radiating one behind the other, and one would almost conclude that the embryonal central vesicles are by this means enabled to retain their own form, the nuclei acting like the keystones of an arch. In addition to this (if karyskinesis is the only way in which cells and nuclei divide themselves in the brain), the conclusion is more certain, that the epithelium of the ventricles represents the matrix for the whole wall of the brain, at least during a considerable period of embryonal life. The same appearances are seen in the retina.

H. Kaan on the Relation between Hypnotism and Cerebral Circulation (Wiesbaden, 1885).—When a warm fomentation is applied to the head of a person in a state of hypnotism, the patient is aroused at once; but with a cold compress, the hypnotic condition is increased. On the other hand, in catalepsy a cold compress produces the lethargic condition. The author concludes from this, that in these two states the conditions of blood supply to the brain are diametrically opposite. He accepts, with Heidenheim, that in the monotonous, prolonged excitation of the senses in the process of hypnotising, there ensues a reflex abnormal anæmia of the brain (lethargy), which, if the excitation be continued, is followed by an equally abnormal hyperæmia (catalepsy). This conclusion is borne out by the variation in the volume of the arm, which was observed by means of Mosso's plethysmograph. In the lethargic stage there was an increase of the volume-curve for the arm (corresponding to the anæmic state of the brain), and in the transition to catalepsy a diminution of this curve was seen. PROF. OBERSTEINER, M.D.

Paneth on the Cortical Motor Area of the Dog (*Archiv f. Physiologie*, p. 523, vol. 37, 1885).—The author's experiments were carried out in the laboratory, and under the supervision of Prof. Exner. He used an apparatus constructed so as to furnish galvanic currents of fixed duration and graduated strength in the exploration of the cortex of narcotised dogs. He first determined the excitable areas with reference to certain muscles, especially those of the feet, one of which—the extensor digitorum, for instance—being selected, and attached to the lever of a myographic cylinder. The corresponding excitable area was next circumscribed by means of an incision, which left it connected with the subjacent parts only. Now if there are any fibres establishing direct communication between the latter and the cortex, the effects of electric excitations must remain the same as before; and this too was found to be the case of course. When such fibres (the direct pyramidal tract) were also divided, no effect was obtained by a moderate current. So far Dr. Paneth confirms in every particular the position held by most physiologists at the present day. The original portion of Dr. Paneth's work is his attempt, by means of circumscribing cuts, to isolate a portion of cortex, stimulation of which should cause movements of one limb or one group of muscles only. This, he expressly states, he failed to do, and as his experiments seem to have been performed carefully, this is an important result. Too much stress must not be laid upon it, however, since a circumscribing cut through the cortex is almost immediately filled with blood, and so spread of the current to neighbouring parts is not prevented. The exact determination of representations of groups of muscles surely is not possible in such an imperfectly evolved cortex as that of the dog, and under any circumstances the problem would be more easily solved by attacking lower and less complicated centres. Dr. Paneth does not appear to be aware of Dr. Mariques' recent work in the same direction, viz. the isolation of motor centres from the neighbouring cortex, which was reviewed in the last number of 'Brain.'

Victor Horsley.

B R A I N.

JULY, 1886.

Original Articles.

A CONTRIBUTION TO THE SYMPTOMATOLOGY OF FRIEDREICH'S DISEASE.

BY JUDSON S. BURY, M.D., B.S. (LOND.),

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The study of the differences between the child and the adult in relation to disease is one of much interest, for although but few morbid processes are special to either period of life, yet certain peculiarities are to be noted as regards frequency, mode of onset and distribution. Our knowledge of the causes of disease is still very scanty, and we find it difficult or impossible to explain why various remote hereditary influences produce their effects some at one age others at another—to say, for example, why pseudo-hypertrophic paralysis originates almost exclusively in early life, or to see a reason for the first attack of gout so frequently occurring between the ages of thirty and thirty-five. Anatomical and physiological phenomena throw much light on some of the pathological tendencies of different ages; it is not very remarkable that the active changes taking place at the growing ends of an infant's bones are so easily perverted, nor is it difficult to trace a connection between the activity of the lymphatics in childhood and the wide dissemination of tubercle at that period.

But in other instances there is a less obvious relation between developmental processes and parts susceptible to disease. It is very difficult, for example, to get a clue to the contrast between the manifestations of rheumatism in the child and in the adult; to understand why implication of joints is usually so slight in the former, especially when we remember how frequent and severe are other joint inflammations in the infant. The problem is very complex, and we need more accurate knowledge of the structure and development of the various parts of the organism; of the time when and the method in which, the various functions become differentiated; of the proclivities of diseases for certain tissues, before we can offer satisfactory explanations of the different morbid susceptibilities of the child and the adult. In the meantime it is important to carefully observe all pathological deviations from adult types, not only to add to our knowledge of the evolution of disease, but in the hope that pathology, as has often happened before, may throw light on physiology.

Turning to diseases of the spinal cord, it may be noted that acute anterior poliomyelitis is common in the infant, rare in the adult; and that the classical type of locomotor ataxy, while one of the commonest of adult nervous diseases, is probably unknown before puberty. But we are scarcely justified in making a more universal assertion as to the relative frequency of affections of the specific elements of the cord at the two periods of life. Both primary lateral sclerosis and amyotrophic lateral sclerosis are most frequently met with in middle life. Seeligmüller[1] observed the typical form of the latter disease in four children of the same family, but without post-mortem proof; while primary uncomplicated lateral sclerosis has not been demonstrated in early life, the evidence in cases of spasmodic paralysis in children pointing rather to primary lesions in the cerebral hemispheres. Our knowledge of affections of the posterior columns in children, though somewhat more advanced, is still fragmentary, and is mainly founded on the observations of Friedreich. Of the five or six cases of primary sclerosis of the columns of Goll, one,

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[1] Seeligmüller, "Sklerose der Seitenstränge des Rückenmarks bei vier Kindern derselben Familie;" 'Deutsche med. Wochenschr.,' 1876, Nro. 16 u. 17.

Du Castel's,[1] occurred in a boy aged 10, and is probably the only case on record of a primary systematic affection of one of the white columns of the cord starting at such an early age. Some seventeen examples of disseminated sclerosis in children are to be found in literature; these, with their two or three autopsies and the more numerous cases of Friedreich's disease, including seven post-mortems, constitute the chief reliable information we have with respect to the rare affections of the special conducting paths of the spinal cord in the child.

The object of the present paper is to bring forward examples of Friedreich's disease, to compare them briefly with the clinical accounts of other recorded cases, and then to study them in relation to sclerosis in patches and to certain aberrant forms.

Edward, 22 years, and his brother, William G., 19 years, were brought to see Dr. Ross at the Royal Infirmary, January 2nd, 1886, and to him I am indebted for an opportunity to examine them. They are the eldest of a family of eleven. The third child, æt. 18 years, had pleurisy some years ago. The 4th, 8th, 9th, and 10th children are quite healthy. The 5th child died of scarlet fever, æt. 5 years; the 6th of convulsions and bronchitis in infancy; the 11th, æt. 10 years, of measles and bronchitis. The 7th child, Albert, æt. 10 years, small for his age, stammers a little in speaking; the head is poked forward in walking, but the gait is normal. Both knee-jerks are completely absent, and the father informs me that Dr. Mackenzie, of Farnworth, also noticed this some months ago. The father and mother, aged 47 and 44 years respectively, have always had good health. The paternal grandfather had small feet, and could not walk well after dark for the last twenty years of his life; he died of a stroke, aged 70; the paternal grandmother of bronchitis, aged 74 years. No other history of disease in the family and none of alcoholism, with the exception of the great-grandfather on the father's side, who drank heavily, but lived to a good old age.

Edward began to walk and talk when 14 months old; he was a long time before he learnt to talk well. Beyond measles in infancy, he was pretty well till 15 or 16 years of age; then he had attacks of dizziness and "uncertain vision, as if he saw double," and it was noticed that his movements were slow. His walk caused merriment; he took too big strides, lifted his foot too high in going up a step; he looked as if uncertain what step to take; he could not regulate his speed, he walked too quickly; his walking gradually became worse, and in about two years' time he was obliged to leave off work every now and then, and for the last eight months has been "completely off his feet." For about one year it has been noticed "that he took things in his hand clumsily, he had no delicate touch at all; anything he got hold of he gripped keen, and it shook very much; he could lift a heavy weight if propped up." His writing has been bad lately; his speech became "thick and strong" about eight months ago, and sometimes he dropped syllables. Has been very torpid for some time, "would sit from one meal to another without stirring." The back has been getting bent the last two or three years. Only quite recently have the head and back been noticed to shake. For two years has been troubled with pains in the legs, chiefly at night; he could not sleep for them, and one night, about two years ago, he cried very hard, the pain was so severe. He passed the 5th Standard when 13, has been a good reader up to lately, and has a good memory.

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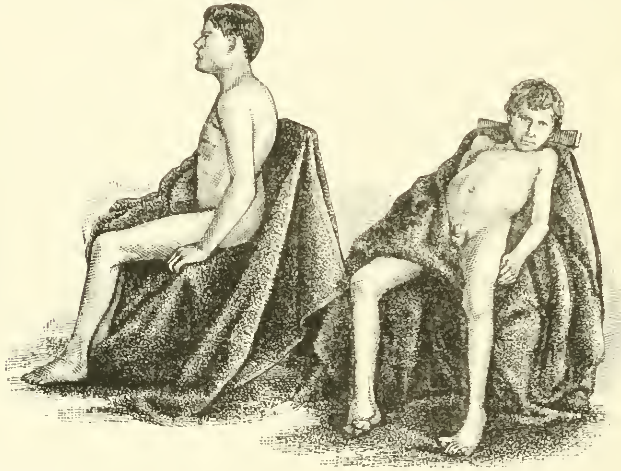
[1] Du Castel, "Observations de sclérose primitive des cordons de Goll;" 'Gaz. méd.,' 1874, No. 3. Soc. de Biol.

Present condition.—Patient looks younger than his age, and has the aspect of a stunted youth of 17 or 18 years. His height is 4 ft. 11 in., his weight 7 st. 6 lbs. He has dark hair and eyes, and a very sallow muddy complexion. There is no wasting; the temperature is normal. He is unable to walk or to stand without assistance. His muscles are well developed; when lying down he can move his limbs freely in all directions, and pretty strongly against passive resistance. The grasp of each hand is good. The knees are inturned and the feet show talipes valgus. When supported, the gait is distinctly ataxic, the legs are thrown hither and thither, and the feet brought down with a stamp. Sometimes the legs are thrown across one another, and this is particularly the case when his eyes are shut; then, too, the feet knock against each other, and he would fall if not supported. The arms and hands can be held out pretty steadily; but in performing complicated movements, such as buttoning the shirt, there is much clumsiness and uncertainty, increased by closing the eyes. On sitting up in bed slight swaying movements of trunk and head are noticeable, but there is no distinct tremor. All the muscles act normally to the faradic and galvanic currents. Patient still suffers occasionally at night from darting pains in the lower limbs. The sensibility of the skin was tested with pin-prick, finger-touch, hot and cold test tubes and the faradic current, and found to be quite normal. He fails to touch the tip of his nose with the end of his finger, and he is not always able to state accurately the position of his legs in bed. Both knee-jerks are completely absent, and the abdominal reflexes somewhat sluggish. The spine presents a sharp lateral curve to the right in the dorsal region and there is extreme lordosis, the somewhat muscular shoulders and upper part of the back standing very prominently behind the level of the sacrum. The special senses are normal. When looking sideways some slight jerks of the eyeballs are seen in a horizontal direction. The tongue is protruded straight and steadily. The upper medium-incisors form a sharp angular notch between them, their free edges appearing to have been chipped off, but they are not pegged, nor otherwise like syphilitic teeth. His voice is like that of a boy at puberty, bass breaking into falsetto; his speech is thick and often difficult to follow, for the words are not distinct, are slurred, and there is a tendency to separate syllables. His head, flat on the top, measures in circumference 21½ inches. Intelligence is very fair; he can converse rationally, and his powers of memory and reflection seem normal; he gives one the impression, however, that a torpor is beginning to steal very gradually over his faculties. When told to look at an object, or to put his head back, he will do so, and maintain the posture for a long time. The chest and abdomen are normal, and the urine is free from albumen and sugar.

His brother William, now aged 19 years, was hearty when a child, and fonder of romping and climbing than his elder brother. Beyond measles when 4 years old, he kept in good health till the present symptoms began to make their appearance when he was 12 or 13 years of age. It was remarked that in walking he turned in his feet, and "he had a way of drawing up his big toes for fun." It was a stumpy walk, as if he were walking on wooden legs, and he brought down his heels with a stamp; it seemed as if the movement came out of the hips, like a bow-legged man. His father looked at his feet to see what made him "walk so queer," and saw that the instep of one foot was quite raised up, and he could put his finger almost under the sole when the boy was standing; one foot was much worse than the other. He got worse, but was able to go to work up to the end of last year. He never complained of pains in his legs like his brother, but felt many a time as if his feet and legs were "drawn up." His arms have been unsteady for three or four years; "he could not carry a glass of water, not because he shook it so much as that he seemed unable to guide it, and also because he often had to run." His speech has been getting worse the last few years, but he was always a broken talker.



EDWARD AND WILLIAM G.—(*From a Photograph kindly taken by my friend,* DR. LARMUTH.)

Present condition.—He has the same sallow, dull, torpid appearance as his brother. His features and his mental condition are also similar. He looks about 14 years old; his height is 4 ft. 7 in.; his weight, 5 st. 1 lb. His head is flat on the top, measures 21¼ inches in circumference, and at the junction at each of the cranial bones there are very prominent bony ridges; behind the lambdoidal suture the occipital bone is much depressed. The special senses, the optic discs, and the activity of the pupils are quite normal. The tongue moves freely, is protruded straight and without tremor. There is slight horizontal nystagmus on tension. The various kinds of sensibility are normally acute. The speech, somewhat monotonous, is in other respects similar to that of his brother. The spine also presents slight lateral curvature and marked lordosis, a plumb line dropped from the cervical region clears the sacrum by nearly 2 inches, and the mid-lumbar region by 4 inches. The feet are much arched; there is marked talipes equino-varus, the extensor tendons stand out prominently on the dorsum of each foot, the toes being drawn up and the great toes extremely so. The muscular power against passive resistance is fairly good. The grasp is feeble; the little fingers are bent at the metacarpo-phalangeal joints. There is slight loss of muscular sense. He can stand with the feet wide apart, but very unsteadily, and not at all with his eyes shut or when his feet are close together. When unassisted, his gait is very staggering. The difficulties of progression seem partly due to the deformity of his feet and partly to ataxia. Standing on the balls of his toes and on the outer edges of his feet, nearly two-thirds of the sole being clear of the ground, he first makes a series of short staggering steps, and having thus secured more equilibrium, he starts to walk; as he rests on the outer edge of the foot of the active leg, the passive leg is thrown quickly forwards and outwards, its foot being brought down with a stamp on the floor, the outer part of the ball of the foot reaching the ground first. Resting on this unsteady and narrowed support, the other foot is raised, and in its turn jerked forwards. The feet are lifted high from the ground chiefly by the contraction of the abductors of the thigh, there being scarcely any flexion at ankle or knee. During progression, which is very hurried, slow walking being almost an impossibility, the head and trunk are poked forwards, the shoulders raised, and the bent elbows stand out from the body like balancing props. The whole aspect of the movement is that of a person staggering hurriedly forwards to catch his balance rather than to make onward progress. Patient says that the floor (which is perfectly level) feels higher in some places than in others, but he has a correct perception of the nature of the surface. With eyes shut, his movements are still more disorderly, and he would soon fall if not caught. The outstretched hands soon begin to waver and deviate from their first position. Fine movements, such as picking up a pin, buttoning his coat, &c., are done clumsily. On sitting up in bed, slight swaying movements of the head and trunk are observed. In getting up from the floor, he first rests on his hands and the tips of his toes; then pushing off with his hands, he throws the weight of the trunk over the legs, his hands catching his knees, and then he staggers into an upright posture or tends to fall backwards. The knee-jerks are lost, the superficial reflexes normal. The upper limbs and thighs act normally to the faradic current, but below the knees the muscles do not contract at all with a weak current, and only feebly and sluggishly with a moderately strong current. The following galvanic reactions were obtained:

	Lower limbs.			Upper limbs.	
	Kat. Cl.	A. Cl.		Kat. Cl.	A. Cl.
Rect. fem..	30	25	Extensors of forearm.	25	25 (slight)
Tib. ant.	30 (slight)	30			
Calf muscles	30 (slight)	30			
Peronei	30 (slight)	30	Biceps	20	20

The clinical history, so similar in the two brothers, accords in almost every particular with that so ably described by Friedreich as distinctive of a group of cases which he separated from ordinary locomotor ataxy on the one side, and from disseminated sclerosis on the other. I have looked through the literature of the subject since the appearance of Friedreich's first paper in 1863, and have been able to collect about 100 cases; but as many of the reports are fragmentary or imperfectly described, some 75 are alone fit for analysis; and though there is not now space to adequately discuss the various clinical and pathological phenomena of this disease,—the less necessary, too, considering the recent clear and excellent Critical Digest by Dr. Ormerod[1] in this magazine,—I propose, before relating other cases, to take a short survey of the main features of other clinical accounts in comparison with those given above.

It has often been pointed out that one of the most striking features of Friedreich's disease is to attack many members of one generation, and there are only four instances, Brousse,[2] Erlenmeyer,[3] Hollis,[4] and Buzzard,[5] where one member only was affected. Ataxia in the ancestry is, however, rare. The family, as Ormerod[6] points out, is often numerically large. There is a proclivity to other nervous diseases, and perhaps especially to alcoholism. In ours the two eldest members of a large family are affected, and in another brother the speech is stammering, and no knee-jerk can be obtained; the latter is also recorded by Ormerod in several members of his ataxic families. The paternal grandfather of our patients had probably an ataxic gait, and similar evidence of transmissibility, though uncommon, is mentioned by Carré,[7] Brousse, Rutimeyer,[8] Ormerod and Massalongo.[9] Of other antecedents, in two of Rutimeyer's the ataxic gait began during convalescence from scarlet fever and typhus; in another it followed chorea; in three of Musso's,[10] small-pox seemed to be the starting-point; and Ormerod also draws attention to the influence of acute disease; in three of his cases the unsteadiness closely followed feverish attacks, and in another case scarlet fever. In Erlenmeyer's, congenital syphilitic leptomeningitis, preceded by five months the symptoms of ataxia : in Smith's,[1] gastric disturbance and palpitation of the heart were premonitory symptoms ; in some others migraine. But in most cases, as in ours, no important antecedent could be discovered. It is a disease of early life, beginning in 75 per cent. between 5 and 15 years, and at an earlier age in boys than girls ; but in ours, at 15 and 12 years respectively : in a few it is stated that the patients never learnt to walk properly.

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[1] Ormerod, J. A., "On the so-called Hereditary Ataxia, first described by Friedreich;" 'Brain,' Vol. VII. p. 105.

[2] Brousse, "De l'ataxie héréditaire." Paris, 1882.

[3] Erlenmeyer, A., "Zur Lehre von den Coordinationsstörungen im Kindesalter;" 'Centralblatt für Nervenh.,' 1883, Nro. 17, p. 385.

[4] Hollis, W. A., "Locomotor Ataxy in a Boy;" 'Brit. Med. Journal,' 1880, ii. 167.

[5] Buzzard, 'Brit. Med. Journal,' 1885, p. 436.

[6] Ormerod, J. A., "An account of two Ataxic Families;" 'Med. Chir. Trans.,' vol. 68, p. 147.

[7] Carré, "De l'Ataxie locomotrice progressive;" 'Thèse de Paris,' 1862.

[8] Rutimeyer, "Ueber hereditäre Ataxie;" Virch. Archiv, Bd. 91, p. 106, 1883.

[9] Massalongo, "Della malattia del Friedreich;" 'Rivista veneta de' Scienze mediche,' 1884.

[10] Musso, "Sulla malattia di Friedreich;" 'Rivista Clinica,' 1884, No. 10.

The symptoms first noticed relate to difficulties in progression, and in more than half, the cases were of an ataxic character, —tottering, staggering, stumbling like a drunken man, are expressions used to describe it ; in other cases weakness or fatigue in walking is mentioned. Sensory disturbances are rarely experienced, but in a few instances pricking or darting pains immediately preceded or accompanied the difficulties in walking. In three or four cases spinal curvature was noted at the onset. In our cases there cannot be a doubt, from the father's accurate description, that the disease began with an ataxic gait in the elder brother, and dizziness was also observed : in the younger it is interesting to note that the drawing up of the great toes was probably present from the first. The movements of the arms did not become incoördinated till some years later, six or seven years in the elder, four or five in the younger brother, and this is the usual course of events ; but perhaps more commonly the spread to the upper limbs is more rapid, and exceptionally the limbs are affected simultaneously. Coming to the fully developed disease, the two brothers present all the characteristic symptoms, namely, incoördination of movement, absence of knee-jerk, curvature of the spinal column, imperfection of speech and nystagmus. With respect to the ataxia, it is usually more pronounced in the legs than in the arms ; it may only show itself in the latter during the performance of complicated movements, or, as in the younger brother, even when the limbs are merely outstretched. From his own cases Friedreich concluded that the incoördination was only exceptionally increased by closing the eyes; but an analysis of all records shows that this holds in about 54 per cent., while the symptom is absent in 45 per cent. In Teissier's[1] case the incoördination was less marked in the lower than in the upper limbs. The slight oscillations of the head and trunk observed in the two brothers are also a frequent phenomenon of Friedreich's disease, and in connection with disseminated sclerosis it is interesting to consider the descriptions of such oscillatory movements. Thus Rutimeyer noted in his fifth and ninth cases frequent jerky shakings of the head; in the former also quiverings of the trunk-muscles even when the body was at rest, and when he held out his arms, jerky tremors of the fingers; in the latter also involuntary tremors of the fingers. In Brousse's, walking was accompanied by tremor of the whole body. In Ormerod's first case, choreic twitchings of the mouth and forehead were seen three years from the commencement of the malady, and four years later, shaking of the head when she tried to write. Erlenmeyer remarked constant movement of the upper limbs; Hammond,[2] in his fifth case, at a late period involuntary twitching of the hand; and Hollis, that there was a constant slow movement of the head from side to side.

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[1] Smith, W. E., "Hereditary or Degenerative Ataxia ;" 'Boston Med. and Surgical Journal,' Oct. 15, 1885.

The ataxia is rarely accompanied by any loss of muscular power, and paralysis, judging from the sixteen cases where it is mentioned, is invariably a late phenomenon; and the same may be said of atrophy of muscles and of contractures noted in some seven or eight cases. Rutimeyer observed in eight of his patients dorsal flexion of the great toes, associated with equino-varus and contractures of some of the fingers; in one, the drawing up of the great toes occurred as early as the fourth year; contractures of the toes, especially of the big ones, are also mentioned by Wälle[3] and by Ormerod. In sixteen cases the electro-muscular contractility is said to be normal; in four, namely two by Carpenter,[4] two by Smith, to be diminished; in the former two the disease, when the electrical examination was made, had existed twelve years; in the latter two, thirty-one and eighteen years. Carpenter simply says that the electric contractility was below normal; Smith, that in one patient the muscular irritability was diminished to faradism and increased to galvanism; in the other patient, that it was diminished to both. There is then only one observation to be found in literature of qualitative alteration of galvanic excitability in Friedreich's disease, and this observation was made on a helpless paraplegic woman, who had first shown ataxic symptoms thirty-one years previously (Case I., Smith). It is therefore of some importance to remember that in the younger of our patients, where the disease had only existed six or seven years, the muscles of both upper and lower limbs showed altered relations to the galvanic current; the anodal closure contraction being obtained more readily than, or at least quite as easily as, the cathodal; and that below the knees there was very decided lessening of irritability to faradism; and associated with this "reaction or partial reaction of degeneration" we have great arching of the feet and contractures of the extensor muscles of the toes.

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[1] Teissier, "Ataxie héréditaire;" 'Lyon médical,' 11 Mai 1884.

[2] Hammond, 'Journal of Nerv. and Mental Disease,' 1882.

[3] Wälle, H., "Zwei neue Fälle von hered. Ataxie;" 'Correspondenzblatt für schweizer Aerzte,' No. 2, 1884.

[4] Carpenter, 'Medical Society of London,' 1870.

As regards the sensory system, shooting or other pains are mostly absent or appear after some years, very rarely at the onset, and are usually slight and temporary. In the first of our cases, in the third of Hammond's, and in the fourth of Ormerod's, the pains are said to have been severe. Diminution of sensibility, principally to touch, has been often recorded, but occurs late; and indeed in two-thirds of the records every variety of sensation was normal. The knee-phenomenon has been found absent in every case examined for it, and in Friedreich's ninth case, within a year after the disease had declared itself; in Gowers'[1] fourth, in Ormerod's fourth, and in Erlenmeyer's, from two to three years after the onset. Its absence in other members of the family not yet ataxic, as observed by Ormerod and myself, indicates that sometimes it may be the earliest sign of the disease; at any rate, it has been clearly established by the investigations of Pelizaeus[2] and others that the knee-jerk is to be always obtained in healthy children.

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[1] Gowers, 'Transactions of Clinical Society,' vol. xiv., 1880, p. 1.

[2] Pelizaeus, "Ueber das Kniephänomen bei Kindern;" 'Arch. für Psychiat.,' Bd. xiv. 1883, p. 402.

A description of the spinal column is given in thirty reports, and in only three was it found normal and after a lapse of nine, thirteen and nineteen years respectively. Lateral curvation was noted in thirteen, a combination of lateral and angular (as in our patients) in twelve, and angular curvature only in two cases. In three instances (Friedreich, Carpenter, and Ormerod) spinal curvature was present at a very early period.

Imperfections of speech similar to those in our cases, and commonly occurring soon after the affection of the arms, are related by most writers; the speech is said to be slurred, stammering, drawling, confluent; and in a few, slow and syllabic; till finally it becomes unintelligible. Exceptionally it would appear to become altered very early, and in Rutimeyer's fifth case it "was always slow and nasal;" and the younger of our patients, according to the father, "was always a broken talker." The movements of the tongue are not impaired, and D'Arcy Power[1] is the only one who mentions a difficulty in mastication and in swallowing. The jerky bilateral movements of the eyeballs on fixation do not commonly appear till the disease has lasted many years; in one case only, the second of Friedreich's, were the jerks *upwards*[2] as well as horizontal. Erlenmeyer notes bilateral nystagmus for three weeks at the onset of the disease, also internal strabismus of the right eye, and twitching of the lower eyelid, and McAlister[3] a slight convergent squint. Mental disturbance is not met with in Friedreich's disease; the intelligence is said to have been weak in D'Arcy Power's and of low type in Brousse's case. Of other symptoms, cold or congested feet, cyanosis and œdema of the lower extremities are noted in a few cases, and also polyuria, salivation and sweating in others, while palpitation, vertigo, and even valvular disease of the heart have been occasionally observed. To complete the clinical picture, it should be added that the march of this disease is very slow, occupying from eight to thirty-three years, but fatally progressive, and that treatment hitherto has been of no avail.

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[1] D'Arcy Power, 'St. Bartholomew's Hospital Reports,' 1882, xviii. p. 305.

[2] Vertical nystagmus is also rare in disseminated sclerosis. See Bouchard, J. B. "Sclér. en plaques nystag. vertical;" 'Journ. de Soc. méd. de Lille,' 1883, v. 321, 332.

[3] McAlister, 'Brit. Med. Journal,' January 2, 1886, p. 19.

Of the morbid anatomy which rests upon seven post-mortems (4 reported by Friedreich, 1 by Schultze,[1] 1 by Brousse, and 1 quite recently by Smith[2] of Massachusetts), we must be content to give the most condensed account. Brousse's patient had been ataxic 8 years; in 2 of Friedreich's, the disease had existed from 12 to 16 years, in the other cases over 20 years. In all the striking and the principal lesions was sclerosis of the posterior columns of the spinal cord, the degeneration being most advanced in the lumbar region, where it was often difficult to distinguish between the columns of Goll and those of Burdach, and least advanced as a rule in the cervical region, and there mainly affecting the columns of Goll. In the cervical region in Friedreich's cases, the lesion was most accentuated at the periphery, in Brousse's it was most intense in the neighbourhood of the central canal and along the edge of the posterior cornua, becoming attentuated towards the periphery. The pia mater at the level of the posterior columns was thickened and pigmented, and fine adhesions were often found between it and the dura mater; in Brousse's case there were no signs of meningitis. In one of Friedreich's cases, the posterior columns were alone affected, but in all the others, changes were found in other parts of the cord; thus in two, the posterior parts of the lateral columns were diseased, in four others not only the lateral but the anterior columns. In Brousse's case the alteration was mainly central, occupying in the lateral columns the angle between the anterior and posterior horns, in the anterior columns the parts adjacent to the anterior horns; in two of the other cases it predominated at the periphery. As to the grey substance it was found quite healthy in only one of the seven autopsies; in one case two canals symmetrically placed one at each junction of the posterior and anterior cornua, mostly within the grey matter but slightly in the lateral columns, extended from the lumbar swelling half-way to the cervical. In two cases there was atrophy of some of the cells of the cornua, in three the cells of Clarke's column were degenerated, and signs of inflammation were present about the central canal. The posterior roots were generally atrophied and indurated. In the cerebrum and cerebellum no trace of chronic disease has been found. Slight changes were observed in the medulla, occupying chiefly the posterior pyramids, and reaching to the level of the nucleus of the hypoglossal, and apparently entirely secondary to the spinal. In one case Friedreich found atrophic changes in the sciatic nerves and in the hypoglossals, and to a less degree in the anterior crurals and in the medians. It should be added that twice the cord and medulla are stated to be smaller than natural, and in Smith's case there was much shrinking of the posterior half of the cord.

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[1] Schultze, 'Virch. Archiv,' Bd. 79, S. 132, 1880.

[2] Smith, 'Boston Med. and Surgical Journal,' Oct. 15, 1885.

From the above brief review of recorded facts it results that we have a homogeneous group of cases closely resembling one another in the principal features of their clinical history, and which it is desirable for the present, at least, to isolate under the name of Friedreich from other affections apparently akin to it. But while doing so for the sake of clearness, it must be admitted that the distinction is based on the general constitution of the malady and not on any particular symptom, and that of this disease, as indeed may doubtless be said of all diseases, there is no absolute pathognomonic sign. It is indeed comparatively easy to find support for the view of Friedreich, Topinard, Erb, Mobius, Carré, and Grasset, that it is a variety of locomotor ataxy, or to furnish reasons for classifying it after Charcot and Bourneville with disseminated sclerosis; for many transitional forms are to be found connecting it with these two diseases, and in trying to form a conception of Friedreich's disease, it is desirable to consider these and other departures from well-known types.

Thus looking at its relation to locomotor ataxia we may first consider Dreschfeld's[1] three cases which were not included in our analysis, the symptoms in the main being those of the classical type of ataxia; thus pain existed from the onset in two of the three, the movements of the arms were free from incoördination after a duration of nine years in the one and fourteen years in the other, and there was neither nystagmus nor alteration of speech. In the first case, after a duration of twenty years it is stated that the patient was "unable to execute any finer movements with his arms, or to grasp anything;" but Dreschfeld, in describing the upper limbs, says "the movements are executed without any incoördination." But incoördination in the uppers, even if distinctly present at such a late stage, is quite consistent with Duchenne's type, whereas every writer testifies to the early occurrence of this symptom in Friedreich's disease. The only resemblance is that five members of the same family were attacked, but it is far better to keep such examples by themselves, calling them instances of family ataxia rather than to class them with Friedreich's disease.

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[1] Dreschfeld (J.), "Family predisposition in Locomotor Ataxy;" 'Manchester and Liverpool Hospital Reports,' vol. iv., 1876, p. 93.

Again, Jakubowitsch[1] gives the following account. A boy æt. 10 years, after suffering six months from symptoms of pachymeningitis, which left complete central deafness, had neuralgic pains in the head, between the shoulders and in the knees, and at the same time weakness of the legs began and the gait became quite ataxic. The lower limbs were slightly atrophied, and the muscular power of both arms and legs diminished; he could only walk when supported, and then ataxicly. There were no contractures. In reading or speaking the boy mixed his words, stumbled and sometimes left out syllables. The patellar reflex were absent on the left, diminished on the right side. The muscular sense and the electro-muscular irritability were lowered. The presence or absence of nystagmus is not stated. The family history was healthy.

The author records it as an example of classical tabes in a child, but I think his description scarcely permits us to say more than that there was some paralysis of the limbs, associated with incoördination in the movements of the lower limbs and with some imperfection of speech. Jakubowitsch glances at the literature of locomotor ataxy in children, and concludes that, omitting Friedreich's cases, there remain probably only six which belong to Duchenne's type, viz. those of Du Castel, Bouchut, Kellog, Kahler and Pick, and Schmid. Now the symptoms of the first were very different from those of tabes and, post-mortem, a pure sclerosis of Goll's columns was proved. Schmid's and Kellog's appear to me rather to be true examples of Friedreich's disease. In the case related by Kahler and Pick,[1] the clinical data are too scanty for classification, and the autopsy revealed degeneration of several tracts of the spinal cord. Bouchut's[2] cases are described as follows: 1. A boy, 11 years old, was suddenly seized with feebleness in the uppers and lowers. He walked badly, had vague pains in the knees and prickings in the feet, but no other sensory disturbances. There was frontal headache, weak sight, occasional diplopia and temporary strabismus; the optic discs were of a pale-rose colour, somewhat flattened and veiled by the congestion. 2. A girl, aged 14 years, affected for two years, who walked slowly and with difficulty, but there was no ataxia. The plantar reflex was gone, but there was no sensory disturbance beyond lancinating pains in the lower limbs and a girdle sensation. There was an occasional momentary embarrassment of speech, diplopia and weak sight, also an excessive hyperæmia of the optic papillæ with a slight infiltration of the retina. We should, in truth, be fairly credulous if on such descriptions we admitted the existence of classical locomotor ataxia in childhood. Nor is there much more satisfactory evidence to be adduced. Of the scattered allusions to the subject none can be deemed wholly conclusive; nor do we know of post-mortems (other than those of Friedreich's disease) made on children who during life had exhibited symptoms of tabes dorsalis. In Cruveilhier[3] we read of a young girl, seized with amaurosis and paralysis, who died of an unknown disease, and the autopsy revealed a degeneration of the posterior columns.

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[1] Jakubowitsch (W.), "Tabes dorsalis im Kindesalter;" 'Arch. für Kinderheilk.' 1883, xii. p. 187.

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[1] Kahler und Pick, "Ueber combinirte Systemerkrankungen des Rückenmarks;" 'Arch. f. Psych.,' Bd. viii., p. 251, 1878.

[2] Bouchut, "Ataxie loc. et sclérose des cordons post. de la moelle chez les enfants;" 'Gaz. des Hôp.,' 1874. No. 38, p. 297.

[3] Cruveilhier, 'Anat. Path.' tom. ii. liv. 32. Paris, 1835-1842.

Bradbury[1] refers to a youth, æt. 18 years, where ataxia followed excessive onanism; and Henoch[2] tells of a boy who, after prolonged irritation of the genitals, was seized with paresis of the lower limbs; he could only walk with help, and then his gait was ataxic, and worse in the dark. There was diminution of the muscular sensibility and lessened activity of the sphincters. He quickly recovered when put under strict surveillance.

Such cases, while throwing but little light on the question of progressive locomotor ataxy ever occurring before puberty, illustrate how difficult it is to explain the phenomena of ataxia. Henoch's case may be assimilated to those of "peripheral nerve tabes," as described by Déjérine,[3] in which, with many of the symptoms of locomotor ataxy, the posterior columns and other parts of the cord were quite healthy, but the cutaneous nerves showed extreme degenerative changes. In this connection the following case, related by Kast,[4] is important. A boy, æt. 14 years, after a slight throat affection (probably diphtheritic), showed disturbances of coördination in the movements of the upper limbs; several weeks later, static ataxia in the legs; finally he could not stand, though individual movements were intact. Marked diminution of sensation in all its varieties was proved, also delay in pain conduction. The patellar reflex was absent; the interossei and the muscles of the tongue were atrophied. Finally, bulbar symptoms appeared, and death from pneumonia took place nine months after the appearance of the first symptoms. At the post-mortem examination the medulla and cord were healthy, but there was a striking degeneration of the peripheral nerves and of the hypoglossi and recurrent laryngeals.

But close as is the apparent relationship between Friedreich's disease and progressive locomotor ataxy, that between the former and cerebro-spinal sclerosis appears to be still closer.

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[1] Bradbury, 'Brit. Med. Journ.,' 1871, p. 565.

[2] Henoch, "Atactische Symptome durch Genitalreizung bei einem siebenjährigen Knaben;" 'Wien. Med. Bl.,' 1880, Nro. 12, 292.

[3] Déjérine, 'La France médicale,' 30th Oct. 1883.

[4] Kast, "Zur pathologischen Anatomie der subacuten Ataxie;" 'Arch. für Psych.,' Band xvi., 1885, p. 865.

In both we meet with imperfection of speech, nystagmus and tremor, unassociated as a rule with either sensory disorders, trophic disturbance or weakness of the sphincters. An analysis of the published cases of disseminated sclerosis in children, brings out the following differences. As a rule sclerosis in patches begins at an earlier age, the mean being from three to four years. Incoördination is the main motor symptom in the one disease, tremor in the other; the gait usually ataxic in Friedreich's disease is spastic in disseminated sclerosis; the tendon reflexes absent in the former are increased in the latter; nystagmus is commonly a more prominent symptom in sclerosis in patches, and strabismus while observed in half the cases is very rare in Friedreich's disease; cerebral disturbance is mostly absent in the latter, and apoplectiform seizures are almost unknown. But considering the various groupings of positive and negative symptoms that have been encountered in sclerosis in patches, the absence for example of nystagmus or of typical speech disturbance, or of tremor on voluntary movement, even the presence of lightning pains and an ataxic gait; taking into account, too, the manifold manifestations of cases regarded as examples of Friedreich's disease, one is tempted to throw all cases of the two diseases into one great group, thereby making the definition of disseminated sclerosis sufficiently wide to embrace not only cases where the lesions are irregularly distributed through the brain and spinal cord, but also those in which certain tracts in the cord are supposed to be more systematically affected. But on the the other hand, whatever may be the exact pathology of Friedreich's disease, and while recognising the transitional forms that seem to take one in a natural sequence to locomotor ataxia on the one side and to disseminated sclerosis on the other, one must admit the striking clinical differences between *typical* instances of the three diseases; and so it is perhaps better, with our present scanty stock of information on the pathology of "systematic" and "diffuse" diseases of the spinal cord, to narrow rather than enlarge the groups of cases embraced by supposed different diseases, to classify anomalies by themselves for a time rather than to include them with typical standards.

Of clinical affinities with disseminated sclerosis, we may mention Seeligmüller's[1] cases. They relate to two brothers and their sister, whose parents were first cousins, and whose mother and maternal aunt were highly neurotic, and the maternal great-grandmother is said to have had club-feet. In the younger brother the symptoms began about puberty, and when seen, aged 26 years, there was static and dynamic ataxia of the lower limbs, and slight incoördination of the uppers. The patellar reflex was unusually active, but the plantar absent. When standing with his feet close together, muscular tremors of the legs were noticed, and marked quiverings of the tendons at the ankles, also tremors of the face when in certain positions, and fibrillary contractions of the protruded tongue. The speech was normal; there was nystagmus, and lateral curvature of the spine. The other brother was affected in a similar manner, having slight strabismus, commencing nystagmus; tremor of the tongue; lordosis; an ataxic gait, but the arms were normal. The patellar reflex was lively, the plantar absent. The speech was peculiar, rapid, and confused, there being rather an ataxia of thoughts than of words. Both brothers were dreamy and forgetful, and the younger brother was also subject to inverted sexual passions. In both the sphincters were uncertain; in neither were there sensory disturbances, paralysis, or contractures. The sister, 21 years old, suffered from nervousness, and it was stated that she could not walk in a straight line. According to Seeligmüller they are unusual cases of Friedreich's disease, but the presence of tremor in connection with exaggeration of the knee-jerk and nystagmus is, I think, a strong reason for drawing them closer to sclerosis in patches. The symptoms of the following patient may in some respects be regarded as complementary to those just related, for while in the latter, there was an association of incoördination of movement with increase of knee-jerk, in the former we see abolition of the knee-jerk in a child who otherwise presents the most characteristic signs of disseminated sclerosis.

Case III.—Rose T. came to Dr. Ross's out-patient department in January 1886, and he with his usual generosity transferred her to my care at the Clinical Hospital. I obtained the following history from her mother. There are two younger children, both quite well. The child's parents are in good health, but the father had "fits" between 23 and 25 years of age. His father and grandparents are alive and well; his mother died of a stroke aged 58 years. The child's maternal grandmother suffers from "cancer of the womb;" the maternal grandfather and the great-grandparents are still living. The uncles and aunts on both sides are free from disease. Rose was a full-time baby, instruments were used, but without injury. She was suckled till 10 months old, was quite well and sensible, and began to walk and talk before a year old. She walked up to two years of age, then one morning she was found to be paralysed down the right side, the face being affected as well as the limbs; this paralysis lasted more than three months. Last November she had convulsions, and was in them from Saturday to Monday; she could not speak after the fits, and the eyes "worked" as they do now, and the mother thinks they "worked" a little before the fits at times; there has been twitching of the hands since the age of two years, which has got worse during the last six months, and for about the same period there has been loss of control over the bladder and rectum. When two years old, she was under the care of Dr. Hutton, at the Children's Hospital, Pendlebury, to whom I am indebted for the following note.

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[1] Seeligmüller, "Hereditäre Ataxie mit Nystagmus;" 'Arch. f. Psych.' Bd. x. S. 222, 1880.

"*January* 26, 1884.—The muscles of the legs are a trifle soft, no obvious wasting. Both legs and thighs are equal in length and circumference. Can stand, but walks as if weak and as though she did not know how to walk. Reflexes very slight, no ankle-clonus, no squint, no facial paralysis, no obvious paralysis of arms. The muscles of the lower limbs react fairly well to the interrupted current. *March* 25. Under the use of massage and the interrupted current, the legs have improved considerably, can now stand and walk a few steps with assistance. Can speak better."

The child's condition at the beginning of this year, then 4½ years old, I found to be as follows—A small child, with bright face, very rapid oscillations of the eyeballs, chiefly horizontal, occasionally rotatory, but never purely vertical; about 140 per minute counting in one direction; the nystagmus ceases during sleep. Pupils and optic discs normal. The head of fair shape measures 19 inches in circumference, the frontal ridge is unduly prominent. She recognises her mother; occasionally asks for a drink; says her name correctly when asked, but enunciation is very indistinct and "scanning;" seems to understand what is said to her, and can name objects held up to her; utters piercing shrieks from time to time. Is unable to raise herself to the sitting posture, but when partly propped up can raise herself a little, can turn her head from side to side, and raise it from the pillow to look through the window, but when she is held up, the head is very apt to fall forward. The head is not affected with tremor, but the limbs shake violently when making voluntary movements. The arms are semiflexed and pronated, and require some force to straighten them. When attempting to grasp, the fingers are widely separated, the first phalanges hyperextended, and the object is clutched violently, and there appears to be a difficulty in relaxing the grasp; the movement is accompanied by violent tremor. When trying to scratch the head, the hand is lifted with much effort and shaking. The lower limbs lie on the bed in rigid extension, the popliteal spaces obliterated, and the tendo Achillis on the stretch. There is slight inversion of the feet, and marked dorsal flexion of the great toes, and to a less degree of the other toes, while the terminal phalanges are slightly flexed; the thighs are in close contact, the adductors being quite rigid. When the thighs are grasped and drawn up, a coarse tremor affects the legs, more noticeable with the right than the left, the former is also a trifle stiffer than the latter. There is no muscular wasting, and the limbs are symmetrical, there being no difference in the measurement of the two sides. When held up erect the lower limbs are rigidly extended and adducted, the tips of the toes just touching the ground; her height then taken is 35 inches. Cutaneous sensibility is normal. None of the superficial reflexes can be got, and the knee, elbow, and radial reflexes are completely absent. The special senses, so far as can be ascertained, are normal. Swallowing appears to be imperfect, there is much noise and gulping in the performance, and a great deal of the milk food is spluttered out of the mouth. The chest is 22 inches in girth, the ribs are slightly beaded, the spine is straight. No signs of disease can be found in the organs of the chest and abdomen. Respiration is rather jerky. I visited the child at home a few days ago (June 4th), and found her greatly wasted, but otherwise she presented the same symptoms, the limbs, however, were stiffer, and I was unable to straighten the tightly-flexed elbows; the knee-jerk could not be elicited; and I should add, that when the patient was in the hospital, repeated trials were made in various positions and by different observers, but no suspicion of knee-jerk was ever obtained.

The association of spastic contractures with loss of the patellar reflex, seems to me very curious; the former indicates disease of the lateral, the latter points to a lesion in the outer half of the posterior columns of the cord in the lumbar region; but, as Westphal has proved, in combined disease of the posterior and lateral columns, rigidity, spastic contractures, and loss of the tendon reflexes do not appear if the disease of the posterior columns reaches down to the lumbar region and involves the posterior root zone, and Ormerod[1] in his analysis of other cases finds this rule to hold. I notice, however, in his abstract of Schultze's second case, that there was "paralysis of lower limbs with rigidity (flexion of knees and thighs, with adduction of thighs); absence of patellar tendon reflex and ankle-clonus;" and post-mortem "fibrillar degeneration" of the posterior columns in their whole length together with extensive sclerosis of the lateral columns. Westphal,[2] too, with the testimony of another case of posterior sclerosis combined with disseminated sclerosis of other parts, extends his dictum by saying that even when there is bilateral rigidity of the quadriceps femoris, the knee-jerk is absent if the disease of the posterior columns reaches down to the lumbar region.

The last cases to be related are also of much interest; they approximate closely to Friedreich's type, and might have been included in our analysis, but are described separately on account of certain peculiarities, the chief of which are: the absence of an ataxic gait, the early onset in the girl of an atrophic paralysis, and the freedom from like disease of other members of the families to which the children respectively belong. For many of the observations I have to thank Drs. Thorburn and Jones, late house-surgeons to the Clinical Hospital.

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[1] Ormerod, J. A., "On the combination of lateral and posterior sclerosis in the spinal cord;" 'BRAIN,' Vol. VIII. p. 110.

[2] Westphal, C., "Ueber strangförmige Degeneration der Hinterstränge mit gleichzeitiger fleckweiser Degeneration des Rückenmarks;" 'Arch. f. Psych.' 1879, ix. Band (xxv. p. 389–412).

Case IV.—John W., now aged 16 years, has been under observation for nearly two years. His mother brought him to the Clinical Hospital in June, 1884, for pains in the sides and legs, and for a shaking of the body; she said that he was quite well till seven years old, when he had scarlet fever and measles; a year later he had a severe fall, cutting the back of his head badly. About this time the present symptoms began to appear, but the mother is uncertain whether after the scarlet fever and measles, or not till after the fall. It was first noticed that he stooped, and that his walking was stumbling and shaky—" he would knock against any one walking by his side." He could not go upstairs without sticking to the banister. He suffered for some time from incontinence of urine. For the last three or four years there has been shaking of the body, his speech has been indistinct, and his temper more irritable.

There are five other children living, all quite well. One child died of croup, another was still-born. The boy's mother had rheumatic fever when a girl, but is now quite healthy. The father died of consumption, aged 34, and his brother also at about the same age. Other relatives on both sides healthy. No history of nervous diseases, or of alcoholism, or of any peculiarity in walking. When admitted to the hospital in July, 1884, he complained of shooting pains which started near the left axilla, passed across the abdomen, and then went down both legs to the toes. He had attacks of these pains several times a day, and said that he had had them for some years, and also a tight feeling round the waist.

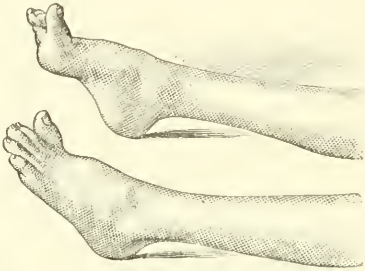
Present Condition.—The boy is fairly grown, being 4ft. 9in. in height. He has a stolid but pleasant expression; his cheeks are red and show slight venous injection. A fine jerky tremor affects the head, the body and the limbs, when these parts are unsupported. Thus the outstretched arms soon begin to oscillate; he can, however, seize a glass of water and carry it to his lips without any perceptible increase of the tremor. When standing, the feet are a little apart, and the hands hang down in front of the body, the head being poked forwards and the shoulders rounded; the balance is not easily maintained, and the tendons of the extensor longus digitorum and of the extensor proprius pollicis are in constant action jerking up the toes. With his feet close together he stands with some difficulty, and when the eyes are closed he totters and falls. He cannot balance himself on his toes, but leaning back does not so easily upset him.

There is much lordosis, the dorsal and lumbar spinal curves being much increased; a plumb-line let fall from the sixth dorsal spine, the most prominent part of the rounded back, falls half-an-inch behind the lower end of the sacrum and $2\frac{1}{2}$ inches behind the most incurved part of the lumbar region. No wasting of the muscles of the back or limbs can be detected, and there is apparently no loss of power; when lying on his back on the floor, with his arms folded across his chest, he can raise himself to the sitting posture. The grasp of each hand is good. As to the lower limbs there is slight knock-knee, the calves are of normal size, and the movements at the various joints are performed with strength, with one exception, namely, flexion at the ankle-joint; this movement is very limited, and during the attempt there is violent dorsal flexion of the toes, especially of the great toe. The feet are remarkable—the plantar arch is greatly increased, and the base of the first metatarsal bone and the internal cuneiform bone form a prominent rounded swelling, from the top of which a slight ridge can be felt, passing downwards and outwards across the dorsum of the foot, between this ridge and the toes there is a deep hollow, to which the hyperextended first phalanges form an almost perpendicular boundary; the other phalanges are flexed. When standing the foot rests on the hardened and prominent balls of the toes and on the heel, no other part of the sole touching the ground, the toes diverging from it at an angle of quite 45°, and beneath the plantar arch the fingers can be passed nearly to the outer border. In walking there is a good deal of general unsteadiness and swaying; the gait does not conform to either the ataxic or the spastic type, the balls of the toes rub against the ground and sometimes trip him up, and the feet are put down heavily, and the toes are constantly being jerked up into extreme extension (dorsal flexion). He leans forward and walks quickly, the pace increasing as if to preserve the balance, which seems to be in constant danger owing to his getting too far forward over his toes. He can walk in a straight line and fairly well with his eyes shut, though with much swaying. No distinct loss of sensation is detectable, yet at the back of the lower part of the thigh and behind the knee a slight touch is not always perceived as well as might be expected. No woolly or other abnormal sensations are experienced by contact of the soles of the feet with the ground in walking. The muscular sense is normal. Picking up a pin brings out a want of coördination in the movements of the fingers. The muscles of the lower limbs while responding readily to faradism show qualitative alterations to galvanism, ACC being obtained with a less current than KCC. As to the reflexes the knee-jerk is completely absent on both sides, and the superficial with the exception of the plantar are somewhat sluggish; indeed, when first examined, it was impossible to obtain the cremasteric; a week or two later this was obtained, but was less active than in health. The special senses are normal, there is no hemiopia and no colour-blindness, memory and intelligence are good, he can read, write, and do mental arithmetic. Slight horizontal nystagmus is seen after a sudden movement of the eyeballs. The pupils are active: the optic discs, though paler than usual, are not diseased. The tongue is protruded straight, and there is a fine oscillation of its fibres. The speech is slow and slightly syllabic. The lungs, heart and abdominal viscera appear quite healthy, and the urine gives normal reactions.

During the boy's stay in the hospital, and afterwards when attending as an out-patient, various modes of treatment were tried but without the slightest effect on his condition. He was carefully re-examined in August, 1885, but no change was observed. To make quite sure of the electrical reactions, I took the patient to Dr. Dixon Mann, who very kindly went over the muscles with me several times, and we assured ourselves that those of the lower limbs undoubtedly presented the partial reaction of degeneration. Thus with the galvanic current the quadriceps contracted at the anodal closure to 4·5 milliampères, but KCC required 12 milliampères, and the tibialis anticus required 10 milliampères for the KCC, but only six for the ACC. Much difficulty was experienced in the examination owing to the involuntary jerkings, not only of the muscle investigated, but of groups of muscles.

When last seen in April this year, patient was much the same; he said he was still subject to occasional darting pains in the lower limbs; he was able to walk quite as well as, if not better than, two years ago.

Case V.—Jane R., æt. 12 years, kindly sent to me by Dr. Ross, was taken into hospital in August 1885, when the following notes were taken. Patient is a delicate-looking thin girl, with a bright intelligent face. Height 4 ft. 3 in.; weight 51 lbs. Temperature normal. She is free from pain. Can sit up in bed without help, but does so unsteadily, and there is distinct oscillation of the unsupported head and trunk, and if, when sitting up she puts her head back, slight jerky movements are observed. The stretched-out arms exhibit slight oscillatory movements. The hands opened as much as possible tend to assume the claw shape, the first phalanges being hyperextended, while the other phalanges remain slightly bent, and cannot by the greatest effort be fully straightened; the extensor tendons stand out over the knuckles, leaving deep hollows between them. Complicated movements are clumsily performed; unbuttoning the dress is difficult, and she can only thread a needle by resting one hand on the other, and steadying her arms against her sides. Both upper and lower limbs are thin and poorly nourished. The feet are much arched, and closely resemble those of Case IV., but the internal cuneiform bone is less prominent, the head of the scaphoid more so, and to it the stretched tendon of the tibialis posticus is seen passing. The extension of the feet at the ankle-joint, their general shape, and the extreme dorsal flexion of the great toes are well seen in the photograph, kindly taken for me by my friend Mr. A. W. Hunton. With a little force the great toes can be straightened, and also the arch of the foot to a slight extent. A thick web joins the second and third toes of each foot for about three-fourths of their length; the third and fourth fingers are also slightly webbed. Flexion of the ankle is a very weak movement. Patient cannot stand erect without assistance; she can, however, for a few seconds, with the feet wide apart, and when leaning forward, and even in that posture, the balance is very insecure as evidenced by the great quivering of the tendons over the front of the ankle and on the dorsum of the foot. When supported she walks in a waddling, tottering fashion, and owing to the dropping of the feet, the legs are lifted high to clear the ground, the balls of the toes, however, often scrape the floor, and the feet are apt to stumble over one another; as the foot rests on the ground, the great toe is also in contact with it, but is quickly jerked up just before the foot is raised. Walking is accompanied by much swaying of the head and body, and the girl is very soon tired. The spine shows a marked lateral curvature to the left, the greatest convexity being opposite the angle of the left scapula; there is also a tendency to lordosis. Cutaneous sensibility appears normal. As regards the muscular sense, she is unable with covered eyes to touch accurately a prescribed spot on the face, nor does she always make correct statements as to the relative position of her legs in bed, nor does she seem so able as a healthy child to appreciate differences between weights tied to the feet. The superficial reflexes are normal,



the knee-jerks quite lost. I am again indebted to Dr. Dixon Mann for much help in the electrical examination. He found the partial reaction of degeneration in the muscles of both arms and legs; thus, with the galvanic current, the flexors of the forearm showed a contraction at the anode when the galvanometer stood at 2 milliampères, but not at the kathode till a deflection of 3·5 milliampères, and in all the muscles of the lower limbs the anodic closure contraction was also much earlier obtained than the kathodic. Nothing wrong was found with the special senses, the intelligence, pupils, or optic discs. Slight lateral tension nystagmus was observed, and also an occasional external strabismus of the right eye. The tongue was steady, the speech drawling, monotonous and indistinct. A loud systolic murmur was heard over the cardiac area, of maximum intensity at the pulmonary cartilage; there was no evidence of disease in the remaining viscera.

The mother states that Jane was a full-time baby, and healthy up to 7 years of age, when it was noticed that she dragged on her right side, leaning towards it in walking; about the same time or soon after her arms became affected, "her hands shook in writing, and things dropped out of them." Her walking became worse, and for the last three or four years she has been unable to walk without help. She began to stammer and speak badly about one year ago, but never used wrong words. She was in the third standard when taken away from school three years ago. Her mother cannot think of any cause for the present disease; the girl never had a fall, or any previous illness, and was never wet through. No other member of the family is afflicted in a similar way; one of her brothers is a complete cripple from infantile paralysis, but with this exception the family on both father's and mother's side is free from nervous diseases.

The known pathology of such cases as the five here related is too limited to enable one to formulate any definite conclusions; but possibly they suggest, when viewed in contrast with classical ataxy, and other chronic nervous diseases of mature life, that there is a greater tendency in the child than in the adult for a lesion, having started in some poorly-developed tract of the spinal cord, to spread with greater speed to other parts, that we may have here a fragment of a more general pathological law applicable to the two periods of life.

BIBLIOGRAPHY.

I. *References to Friedreich's Disease, and to Ataxia in Childhood.*

Carré: "De l'Ataxie locomotrice progressive."—Thèse de Paris, 1862.

Friedreich: "Ueber degenerative Atrophie der spinalen Hinterstränge."—Virch. Archiv, Bd. 26, S. 391, 433; Bd. 27, S. 1. 1863.

Topinard: "De l'Ataxie locomotrice."—Paris, 1864.

Carré: "Nouv. recherches sur l'ataxie locomotrice."—Paris, 1865.

Bradbury: 'Brit. Med. Journal,' 1871, p. 565.

A. Carpenter: "Report of Medical Society of London."—'Lancet,' p. 779, 1871.

Bouchut: "Ataxie loc. et sclérose des cordons post. de la moelle chez les enfants."—'Gaz. des Hôp.,' 1874. No. 38, p. 297.

Kellog: "Two Cases of Locomotor Ataxia in Children."—'Archives of Electrol. and Neurol.,' vol. ii., p. 182, 1875.

Hitzig: "Saw three children, 5–10 years, of a tabetic mother, whose gait was disordered."—'Berl. Klin. Wochenschr.,' 1875, p. 513.

Friedreich: "Ueber statistische Ataxie und atáktischen Nystagmus."—'Arch. f. Psych.,' 1876, vii., 235–238.

Friedreich: "Ueber Ataxie mit besonderer Berücksichtigung der hereditären Formen."—Virch. Archiv, Bd. 68, S. 145; Bd. 70, S. 140. 1876–77.

Dreschfeld: "Family Predisposition in Locomotor Ataxy."—'Manchester and Liverpool Hospital Reports,' vol. iv. 1876, p. 93.

Shaw (J. C.): "A Case of Ataxia in a Child."—'Tr. Am. Neurol. Assoc., N.Y.,' 1877. ii. 85–87.

Vulpian: 'Maladies du Syst. Nerv.,' p. 245, 1877.

Kahler und Pick: "Ueber combinirte Systemerkrankungen des Rückenmarks."—'Arch. f. Psych. und Nervenk.,' Bd. viii., 1878.

Erb: "Krankheiten des Rückenmarks."—'Handbuch der speciellen Path. und Therapie von Ziemssen,' Bd. xi., 1878.

Eulenburg: 'Lehrbuch der Nervenkrankheiten,' Bd. ii., S. 458, 1878.

Möbius: "Ueber die hereditären Nervenkrankheiten."—'Sammlung klinischer Vorträge von Volkmann,' No. 171, 1879.

Friedrich Schultze: "Ueber combinirte Strangdegenerationen in der Medulla spinalis."—Virch. Archiv, Bd. 79, S. 132, 1880. (He gives here the morbid anatomy of one of Friedreich's cases.)

Seeligmüller: "Hereditäre Ataxie mit Nystagmus."—'Arch. f. Psych. und Nervenk.,' Bd. x., S. 222, 1880.

Seeligmüller: "Sclerose der Hinterstränge."—'Handbuch der Kinderkrankheiten von Gerhardt,' 1880, S. 187.

Henoch: "Ataktische Symptome durch Genitalreizung bei einem siebenjährigen Knaben."—'Wein. Med. Bl.,' 1880, No. 12, 292.

Schmid: "Ueber hereditäre Ataxie."—'Correspondenzblatt f. schweizer Aerzte,' Bd. iv., S. 97, 1880.

Gowers: "Trans. Clin. Society," vol. xiv., p. 1, 1880.

Hollis (W. A.): "Locomotor Ataxy in a Boy."—'Brit. Med. Journal,' 1880, ii., 167.

Grasset: 'Traité pratique des maladies du système nerveux,' Paris, 1881, p. 321.

Brousse: 'De l'ataxie héréditaire,' Paris, 1882.

Féré: "Maladie de Friedreich."—'Progrès Médical,' x., p. 890, 1882.

Davidson: "Two Cases of Ataxia in Children."—'Med. Times and Gazette,' April 22, 1882.

Aur. Bianchi: "La malattia del Friedreich."—'Gazzetta degli Ospitali,' N. 95, 1882.

Hammond: "On the so-called Family or Hereditary form of Locomotor Ataxy."—'Journal of Nerv. and Ment. Disease," 1882, p. 484.

Leubuscher: "Ein Fall von Tabes dorsalis im frühesten Kindesalter."—'Berl. Klin. Wochenschr.,' 1882, No. 39.

D'Arcy Power: "A Case of Hereditary Locomotor Ataxy."—'St. Bartholomew's Hosp. Reports,' 1882, xviii., 305–308.

W. (F.): "Diffuse sclerosis of the Spinal Cord and Medulla Oblongata."—'Disease of Friedreich, Phil. M. Times,' 1882–1883, xiii., 372–375.

Leading article on "Friedreich's disease."—'Brit. Med. Journal,' 1883, i., 627.

Erlenmeyer: "Zur Lehre von den Coordinationsstörungen im Kindesalter."—'Centralblatt für Nervenk.,' 1883, N. 17, S. 385.

Rüimeyer: "Ueber hereditäre Ataxie."—Virch. Archiv, Bd. 91, S. 106, 1883.

Seppilli : "Atassia ereditaria, o malattia di Friedreich."—'Rivista sperimentale di Frenatria,' 1883, p. 335. (Contains an account of the literature.)

Jakubowitsch : "Tabes dorsalis im Kindesalter."—'Arch. f. Kinderheilk.,' 1883, xii., 187.

Massalongo : "Della malattia del Friedreich."—'Rivista Veneta di Scienze mediche,' 1884 (with photographs).

Musso : "Sulla malattia di Friedreich."—'Rivista Clinica,' 1884 (giving the German literature).

Teissier : "Ataxie héréd. avec dégénérescence des faisceaux post. de la moelle."—'Lyon Méd.,' No. 19, 1884.

Longuet : "Critical digest of Friedreich's disease."—'Union Médicale,' 18 Mai 1884.

Wälle (H.) : "Zwei neue Fälle von hereditärer Ataxie."—'Correspondenzblatt f. schweizer Aerzte,' No. 2, 1884.

Ormerod (J. A.) : "On the so-called Hereditary Ataxia, first described by Friedreich. A critical digest."—'Brain,' Vol. VII., p. 105, 1884–1885.

Seguin (E. C.) : "Clinical Report of two sets Friedreich's Hereditary Ataxy."—'Medical Record,' New York, 1885, xxvii., 645–647.

Palma : "Malattia del Friedreich."—'Relazione sulla cura delle acque del Gurgitello praticata in Napoli,' 1885.

Fazio (G.) : "Un altro caso della cosiddetta atassia ereditaria o tabe del Friedreich."—'Rivista Clinica e Terapeutica,' p. 73, 1885.

Vizioli : 'La malattia di Friedreich,' Naples, 1885.

Remak : "Three Cases of Tabes dorsalis in Children."—'Berl. Klin. Wochenschr.,' No. 7, 1885.

Raymond : "Tabes dorsalis et Tabes spasmodique."—'Dict. Encyclop. des Sciences Médicales,' Paris, 1885.

Botkin (J.) : "Sluchai Fridriecho voi bolezni."—'Med. Oboz. Mosk.,' 1885, xxiii., 32–38.

Smith (W. E.) : "Hered. or Degenerative Ataxia."—'Boston Med. and Surgical Journal,' Oct. 15, 1885 (with photographs).

Ormerod (J. A.) : "An Account of two Ataxic Families."—'Med. Chir. Trans.,' vol. 68, p. 147.

McAlister : 'Brit. Med. Journal,' January 2, 1886, p. 19.

II. *References to published Cases of Disseminated Sclerosis in Children are given by Marie and Moncorvo.*

MARIE (P.): "De la sclérose en plaques chez les enfants."—'Revue de Médecine,' 1883, p. 536–568.

MONCORVO: "De la sclérose en plaques chez les enfants."—'União Med.,' Rio de Jan., 1884, iv., 16, 49, 97, 145.

STADTHAGEN: "Herdformige Sclerose nach Diphtherie."—'Arch. f. Kinderheilk.,' 1883, v. Bd., S. 1.

PELIZAEUS: "Ueber eine eigenthümliche Form spastischer Lähmung mit Cerebralerscheinungen auf hereditärer Grundlage."—'Multiple Sclerose, Arch. f. Psych.,' xvi. Bd., p. 698, 1885.

III. *For references to and analysis of cases of Combined Sclerosis of the Posterior and Lateral Columns, see* Ormerod's Critical Digest, 'BRAIN,' April 1885.

ON SOME OF THE RARER FORMS OF MUSCULAR ATROPHIES.

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(*Continued from page* 190, *July*, 1885.)

CASE V.

L. B., æt. 55, married, housekeeper, was admitted into the Infirmary on July 20th, 1883, and gave the following history: She had been well till twelve months before admission, when she began to experience a weakness in her ankles, with "twitching sensations," the feet also became numb, and from time to time she noticed slight swelling and redness of the top of each foot; the weakness increased, and she was at last obliged to lie up; about two months before admission, when trying to walk, she fell; very soon after this her hands became weaker and thinner, and in the course of four weeks she completely lost the use of them.

On admission the patient was found to be a person of middle stature and healthy looking; her face, trunk, and upper arms were very fairly nourished, but the hands and forearms were very markedly atrophied. The digestive, circulatory and respiratory systems were normal; bladder and rectum unaffected. The nervous system was the only one which showed marked changes.

The patient was unable to walk or stand; the hands and forearms, besides being atrophied were paralysed; the movements of the shoulder were somewhat limited; those of neck and head were perfectly free.

Lower extremities: the muscles, both of the right and left leg showed some atrophy and felt flabby; the patient was unable to perform any movement with the toes or feet; the muscles of the thighs were less wasted, and the patient was just able to flex and extend the knee slightly, and also the hip; she was able also to abduct and adduct the leg slightly; she was not able to move from the recumbent posture without assistance. There was some slight swelling of the dorsum of the foot. There was marked anæsthesia of the soles of the feet and of anterior and inner part of both legs, more marked on the left than on the right side; the sense for pain and the sense for temperature were much diminished on the inner side of both legs; the thighs showed no anæsthesia, but on the contrary seemed rather hyperæsthetic and sensitive to pain, and differences of temperature were at once perceived in any part of either thigh.

The superficial reflexes were diminished and the deep reflexes entirely absent.

MEASUREMENTS:

	RIGHT.	LEFT.
Calf	10¾ inch.	10½ inch.
Instep	8¼ ,,	8¼ ,,
Middle of thigh	15 ,,	14½ ,,

The electric reactions of the muscles were tried several times, and it was found that the calf-muscles, the peronei and the extensors long. digit. were only made to contract with very strong faradic currents, whilst with the galvanic current they showed A C C > K C C.

Upper extremities: there was very marked atrophy of the interossei muscles and the muscles of the thenar and hypothenar eminences, with complete loss of power; the fingers were in a slightly flexed position, the patient was unable to flex them further or to extend them; she was likewise unable to flex or extend the wrist of either side, the muscles of each forearm were flaccid and atrophied, and pronation as well as supination was impossible; flexion and extension of the elbow, though much impaired, could be carried out. The shoulder muscles appeared well nourished, and on the right side all the movements were fairly normal, on the left side abduction of the humerus was impaired.

There was some anæsthesia both in the palm and back of each hand, with marked loss for pain, whilst the sense for temperature seemed intact. On the forearm and arm sensation was normal, except over some few patches on the inner side, where the skin appeared hyperæsthetic, the sense for pain and for thermal differences appeared normal over the upper part of each forearm. The sensibilities of the arms and shoulders were normal. The muscular sense is normal. The deep reflexes (over the radius and triceps) were absent. There was some slight œdema, without redness, over the back of each hand.

MEASUREMENTS:

	RIGHT.	LEFT.
Over middle of biceps	8¼ inch.	8½ inch.
Middle of forearm ..	7 ,,	7½ ,,
Dorsum of hand ..	6¾ ,,	7¼ ,,

It will thus be seen that while in the lower extremities the muscles were more atrophied on the left side, the right upper extremity showed more atrophy than the left.

On testing the electrical reactions, results were obtained similar to those in the lower extremities. To the faradic current, some of the atrophied muscles did not respond at all, others only when the secondary spiral was approached to 40 mm.; unaffected muscles responded when the two spirals were at a distance of 110 mm., some when they were 120 mm. distant.

With the galvanic current the affected muscles showed A C C > KCC, and thus with the Dixon-Mann battery the following results were obtained:

Flexors of the forearm:—

LEFT.		RIGHT.	
KCC	30 cells.	KCC	35 cells.
ACC	25 ,,	ACC	30 ,,
AOC	40 ,,	AOC	40 ,,

Extensors of the forearm:—

KCC	20 cells.	KCC	25 ,,
ACC	20 ,,	ACC	20 ,,
AOC	30 ,,	AOC	30 ,,

The muscles of the trunk were weak and the patient was not able to raise herself in bed; the thorax, however, was well expanded with every inspiration and the diaphragm acted well: there was no pain or tenderness over the spine along its whole length.

The sensibility for tactile impressions, for pain and for temperature was normal over the front and back of the chest and abdomen. The superficial abdominal and thoracic reflexes were almost altogether absent.

The muscles of the neck showed no impairment of either motion or sensibility.

Examination of the cerebral nerves revealed nothing abnormal.

The pupils were equal and reacted well to light and accommodation. Fundus oculi normal on both sides. The other special sense organs were normal, and there were no cerebral symptoms, such as headache, vertigo, &c.

Urine has s. gr. 1022, contained neither sugar nor albumen.

The temperature was a little below the normal, and the patient perspired on both sides equally.

The patient remained in the hospital till the beginning of December; she was treated with the galvanic current, and took various drugs, such as bromide of potassium, iodide of potassium, and strychnia; massage was also practised—but the symptoms remained almost unaltered.

The exact diagnosis presented some difficulties. Briefly recapitulated, the chief symptoms were an almost complete paraplegia of the lower extremities, with some atrophy of the muscles of the foot and very marked symmetrical atrophy of the muscles of the hands, fingers, and forearms, the atrophy being however, more pronounced in the left foot and the right hand and forearm; there were also disturbances of tactile sensibility, and of sensibility for pain of the affected parts. The sense for temperature was impaired only over the affected muscles of the lower extremities. The muscular sense was normal.

The tendon reflexes were absent.

The bladder and rectum were unaffected.

There was some œdema of the backs of feet and hands.

The disease came on somewhat slowly in the lower extremities, but the atrophy in the hands and fingers, according to the patient's statement, came on very rapidly.

In many points the case resembled one of syringomyelia, especially as we had the altered sensibilities, which, according to Schulze, Remak, and others, are constantly found in that disease along with the trophic changes; on the other hand, the paraplegia pointed more to an extensive myelitis of the lumbar region.

By her own wish the patient left the hospital, and I had no opportunity of again seeing her alive. Soon after she had reached her home she began to suffer from bedsores, and she died somewhat suddenly on January 6th, 1884.

I was not allowed to examine anything but the spinal cord, and was prevented from taking away any part of this for further examination; and I have therefore to regret very much that the post-mortem account is so incomplete.

The inspection showed no affection of either the bones or the membranes. On taking out the cord, some parts, especially the lumbar and cervical enlargements, felt soft. Transverse sections showed in the lumbar region a soft, almost diffluent central part, reddish in colour, round which the peripheric portion of the cord formed a sort of envelope or sheath; the softening extended considerably into the anterior and posterior grey matter. The softening could be traced up for about an inch when it gradually tapered off; the lower dorsal region of the cord was very soft, but showed no discoloration, nor any marked change of consistency between the central and peripheric parts; the upper dorsal region was also found softer than normal, but the central canal and the grey and white matter could be made out easily. The lower cervical region, on the other hand, showed again marked central softening like the lumbar part; the softened part had a dark red appearance, it involved the central canal, and the adjacent anterior and posterior grey matter; at no place did it reach the peripheric white matter, which here too formed a sort of mantle round the altered mass; the upper part of the cervical cord appeared normal. The medulla and brain could not be examined.

From this incomplete inspection, made under the most unfavourable circumstances, it appears that we had here two foci of central myelitis, one in the lumbar and one in the cervical region; how far the intervening portion of the cord was affected, I am unable to say. That the lesion was inflammatory and not a simple hæmorrhage, as one might suppose from the colour of the softened mass, is, I think, evident from the slow onset, the symmetrical situation of the symptoms of the lesion, and from the absence of any signs of improvement, which almost invariably follow hæmorrhage into the cord, if this be not fatal within a short time of its occurrence. For in the cases of hæmorrhage, absorption of some of the extravasated blood takes place, and those parts which are simply compressed and not actually destroyed by the hæmorrhage, regain their function. Thus in a case of hæmatomyelia of the cervical region recently under my care, and briefly reported,[1] there was almost complete paraplegia of the lower extremities and one upper extremity, the patient regained power over the lower extremities, and to a great extent of the affected upper extremity, and there remained only an atrophy of some muscles of the shoulder and arm.

I look therefore upon the case as one of central myelitis. The symptoms observed were quite in harmony with the lesions found after death.

It would extend the paper much beyond its scope were I to relate the cases of tumour of the spinal cord, or cases of hæmorrhages into the cord, which I have observed during the last few years, and where muscular atrophy formed a principal symptom; such cases present as a rule no difficulty in the diagnosis, and can be easily distinguished from those atrophies where the ganglia cells are primarily affected. One case, however, I will very briefly allude to, namely, a case of tubercular tumour in the lumbar portion of the spinal cord.

CASE VI.

R. H., æt. 19, was admitted into the infirmary July 21, 1883, suffering from ascites. The patient had enjoyed good health till a few weeks before admission, when he noticed the abdomen increasing in size; the distension became so great that he sought admission into the Infirmary. His habits had been most temperate, and there was no evidence of any syphilis.

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[1] 'Brit. Med. Journ.' 1885, vol. ii.

On admission the patient's abdomen was found very much distended (girth of abdomen forty inches), with fluid running freely in the cavity ; the liver dulness could not be determined ; the spleen seemed pushed up, but not enlarged ; there was no enlargement of the superficial abdominal veins ; the feet were slightly œdematous. Beyond this nothing further could be made out on physical examination. The respiratory, circulatory, and nervous systems were perfectly normal. The patient looked anæmic and thin, and in the absence of alcoholism it was difficult to account for the ascites, especially as there was no pain and no rise of temperature to indicate any peritonitis. The urine was free from albumen.

The case was looked upon as either one of those cases of pure ascites described by G. Johnson, or possibly of tubercular peritonitis, for in the latter there may be little pain, no rise of temperature, and a large quantity of exudation, which in character resembles a serous transudation.

On July 24, the patient was tapped, and 18 pints of straw-coloured, thin serum were withdrawn. Unfortunately no part of the fluid was saved, to allow us to look for tubercle bacilli in the evacuated fluid. The girth of abdomen after tapping measured only thirty-one inches. The abdomen was examined after the tapping, but no tumor could be detected ; the liver did not reach beyond the line of ribs.

For a time the patient seemed better, the fluid did not reaccumulate ; the œdema of the feet disappeared, and the pulse, which had always been above 90, became slower and stronger.

On August 7th, the patient complained of a feeling of cold in the right leg and foot, and shooting pains down the right leg ; soon afterwards the left leg became the seat of pain. This was followed by weakness which rapidly increased, so that on August 18, both legs were almost totally paralysed—the right more than the left ; the legs could not be moved from the bed ; adduction, abduction, and rotation of the legs was lost on both sides. The tendon reflexes had disappeared. The paralysed muscles still responded normally both to the faradic and galvanic current. The sensibilities were found but little impaired, except on the right foot where there was anæsthesia, and the cutaneous reflexes appeared normal.

The paralysis after a time improved, and the patient was able to flex the thigh on the abdomen, and the leg on the thigh; he could also rotate the legs slightly inwards, but not at all outwards. There was, however, now marked atrophy and paralysis of the extensor femoris each side, also of the gluteal muscles on each side. The atrophied muscles only responded to very strong faradic currents; with the galvanic current they did not show degenerative reaction.

The general condition of the patient became worse; the ascites did not reappear; and there were no symptoms pointing to any affection of the lungs, but the patient lost flesh somewhat rapidly and suffered much from headache.

On Sept. 3rd the patient had several severe general convulsions. During the month of September the patient had several more convulsions, and complained of pain in the neck and back; the upper extremities remained normal; the condition of the lower extremities improved, with the exception of the atrophied muscles. The tendon reflexes remained absent. The chest organs and abdominal organs showed no further symptoms; there was no diarrhœa, and the patient had complete control over his bladder and rectum. There were profuse night-sweats, and the temperature, which had been subnormal on admission and had only been slightly above normal for some weeks, now ran up to 102° and 103° in the evening.

The condition of the patient underwent but little change during the month of October, except that the general emaciation increased, and his appetite, which had been fairly good, now failed him. The patient became somnolent on October 29th; the pupils, which had reacted well, were found dilated, and did not react to light; the fundus of the eye, however, was normal; pulse 94, respirations 36, temperature 103·2°; there was some vomiting. The patient moaned a good deal. On Oct. 30th, the patient became comatose, the breathing became stertorous, and he died the same day.

The clinical diagnosis—to wit, tubercular peritonitis, tubercular cerebro-spinal meningitis, and a tubercular tumor in the lowest portion of the lumbar cord, was verified by the post-mortem examination made by Dr. Maguire.

The lungs were healthy, the abdominal cavity contained no fluid, but the intestines were matted together by numerous adhesions, and the omentum and peritoneum were thickened and studded over with numerous tubercles; the intestines were healthy.

On opening the spinal canal the dura mater was found thickened, the pia mater thickened and studded over with numerous little white nodules, which proved to be tubercles; the cord showed in the lowest portion of the lumbar region, close to the corda equina, a firm white tumor, situated centrally and extending laterally near, but not quite up to the periphery. Microscopic examination of the tumor showed it to be composed of a number of small tubercles with very large and well-formed giant-cells. The rest of the cord appeared quite healthy. Examination of the hardened cord showed marked ascending degeneration which in the lumbar region affected both Goll's and Burdach's tract, whilst in the dorsal and cervical region the degeneration was limited to Goll's tract only.

The pia mater of the brain showed numerous miliary tubercles, both at the base of the brain and in the sylvian fissure; the brain appeared normal, the ventricles were much distended.

I have referred to this case because Pitres and Vaillard[1] have recently described cases of peripheric neuritis with marked atrophy of muscles, complicating tuberculosis. The existence of such peripheric neuritis has, therefore, to be borne in mind, when paralysis and atrophies appear in persons suffering from phthisis. Though we did not examine the peripheric nerves in our case, yet there can be no doubt that the symptoms were due to the tumor in the lumbar portion of the cord; this was particularly shown by the electric reactions and the presence of other spinal symptoms, such as the complete paraplegia which characterised the onset.

The *second* group of muscular atrophies includes those cases where the affection is one of the peripheric and intermuscular motor nerves. This class of cases is just now attracting the attention of many neurologists, and peripheric neuritis is found extensively distributed. Besides the multiple peripheric neuritis of Leyden, we see peripheric neuritis following acute zymotic diseases, chronic intoxications, such as those caused by lead, alcohol, arsenic, and chronic diseases such as phthisis and diabetes. Quite recently Dr. Morgan and Dr. Ross showed some marked cases of peripheric neuritis before the Manchester Medical Society, with atrophy produced by the inhalation of bisulphide of carbon vapour. The muscular atrophy which is seen in some of these neurites is very marked, as *e.g.* in alcoholic paralysis, or in some cases of lead paralysis.

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[1] 'Revue de Méd.' 1886, No. 3.

Cases of alcoholic paralysis with marked muscular atrophies I have already described,[1] and need not therefore refer to these again.

I beg leave, however, to relate briefly two cases, which seem to me to belong to the class of peripheric paralyses, but where there was marked atrophy.

CASE VII.

T. W., æt. 43, was admitted into the Infirmary under my colleague, Dr. Simpson, Feb. 14, 1882, suffering from paralysis and atrophy of both upper extremities, which had only been coming on a short time. The patient stated that she had been married 23 years, and had had 12 children, of whom 9 were still living; her occupation before she was married was that of a "cotton-baller," but she had not been working since her marriage. Three weeks before admission she felt a quick and shooting pain across her right hand; the pain was especially severe over the little and ring fingers; very shortly after this the wrist dropped, and soon she found her arm also paralysed, the shoulder being more affected than the elbow; the whole arm was very painful. Fourteen days later the left arm became attacked, here also pain in the ring and little fingers was the first symptom, and this was followed by wrist drop and paralysis of the arm and shoulder, with great pain in the affected part, and pain across the shoulders from side to side.

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[1] 'BRAIN,' Jan. 1886.

I had an opportunity of seeing and examining the patient during her stay in the Infirmary, and found her suffering from marked atrophy and paralysis of the muscles of the fingers and hand; there was also marked paralysis and atrophy of the extensors of the fingers and wrist; the flexors of the fingers and wrist were also affected, but not to such a great degree as the extensors; the muscles of the arm were likewise affected, the deltoid, the biceps and the supinator longus less than the triceps—but all movements were very much impeded—the patient could not raise her arms or adduct them, extension and flexion of the elbow was carried out only very feebly, and extension and flexion of wrist or fingers was quite impossible.

The patient complained of numbness and coldness of her fingers, but no distinct anæsthesia could be made out. The paralysed and atrophied muscles showed marked degenerative reactions.

During May, 1882, the patient came under me as an out-patient and I found her somewhat better, but there was still marked paralysis and atrophy of most of the muscles; after a time she improved, and eventually became perfectly well.

In January, 1884, the patient presented herself again in the Out-patients' Department, suffering from a very similar but not quite so far advanced attack of paralysis with atrophy. The attack was again ushered in by pain in the fingers, soon followed by paralysis of the muscles of the fingers and hands; the flexors of the hands and the flexors of the arm were considerably less affected than the extensors. There was again found marked degenerative reaction in the affected muscles. After a few months' treatment the patient completely recovered.

In March, 1885, the patient again came to the Out-patients' Department, suffering from a third attack, but of a milder type. The patient complained of some pain in her fingers, and there was some paresis of the extensors of the wrist and fingers of each upper extremity; but this time the affection yielded soon to treatment, and the patient, whom I saw quite recently, has had no other attack since.

(The sister of the patient has been under the care of Dr. Ross, and has had similar attacks, seven in number; her case was looked upon by Dr. Ross[1] as one of Syringomyelia, and I have Dr. Ross's authority for stating that he now believes that his case, like mine, was really a case of peripheric neuritis.)

There was no suspicion of alcoholism in this case, nor anything pointing to lead-intoxication—and I therefore look upon it as a case of so-called rheumatic peripheral neuritis; the onset, the pain, the electric reactions and the complete recovery all point to this as the only likely diagnosis. Interesting are the repeated attacks, and the fact that her sister had suffered from a somewhat similar affection.

Case VIII.

J. M., æt. 28, single, was admitted, June 15th, 1885, into the Infirmary under Dr. Morgan. I had an opportunity of seeing the case several times. I am indebted to Dr. Morgan for his kindness in allowing me to publish the case, and to Dr. Bury, the medical registrar of the Infirmary, for the notes of the case.

The patient, a packer by trade, had good health before present illness, though his work compels him to be in a hot, close and dark room; he had been always temperate and never had syphilis. About a month before admission he complained of a good deal of general pain. On May 26th he was going up some steps, when the right leg suddenly gave way from pain; he was able afterwards to walk again, the pain reappeared, and he was only able to take very short steps when walking. Soon after he was not able to walk at all, and was obliged to take to his bed, and was not able to raise himself. He next noticed that his legs became drawn up and he had great difficulty in straightening them, and at last they remained flexed. A fortnight before admission he also commenced with pain in his right chest, difficulty of breathing and palpitation.

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[1] 'Nervous Diseases,' 2nd ed., vol. i., p. 934.

State on admission: Patient has fair complexion and is much wasted; he lies on his back with his legs drawn up; the knee can be extended to an obtuse angle, but any further extension gives great pain and chiefly in the flexor muscles at the back of the thigh, at the same time, fibrillar contractions of these muscles are seen; he can flex and extend both feet, but inward and outward movements of the feet are very slight, especially on the left side. He can abduct and adduct the thighs with strength, and can also flex and extend the thighs.

Sensation to touch and pain in the lower extremities is normal; the patient however does not always discriminate between a very slight touch and a very slight prick; the muscular sense is normal; the knee jerk is absent. Slight pressure on the calves gives rise to great pain. Electric reaction with constant current:

	Right.		Left.	
Tib. antic.	KCC	25	KCC	25
and ext. com. ..	ACC	25	ACC	20
	AOC	30		
	KOC	30		

	Right.		Left.
Tibial post.	KCC	25	45
	ACC	30	35
Rectus femoris ..	KCC	45	50
	ACC	35	35
	AOC	50	50

The upper extremities are much wasted, but the muscles of right arm and forearm are fairly strong, while the flexors and extensors of left forearm are weak.

Dynamometer in right hand marks 45 Kgm.
„ left „ „ 20 „

The extensors of the fingers and wrist are weak on both sides, but most markedly so on the left side; the thenar and hypothenar eminences are much wasted; the interossei appear normal.

The muscles of arm and shoulder are normal on both sides. The sensation both to touch and to pain appears normal in both upper extremities; there is, however, some slight muscular tenderness on pressure.

The electric reactions of muscles of upper extremities show again degenerative reaction, as seen from the following figures :—

Extensors of fingers :—

Right.		Left.	
ACC	15	ACC	15
KCC	20	KCC	20
AOC	25	AOC	15

Flexors of fingers :—

Right.		Left.	
ACC	10	ACC	10
KCC	15	KCC	15
AOC	15	AOC	10
KOC	35		

The patient complains of no pain in the back. The cranial nerves and special sense organs are normal.

Examination of the chest shows dulness on percussion on the right side behind, with diminished breath-sounds, and absence of fremitus. The apex of right lung and the whole of left lung appear normal.

On June 23, a little clear serous fluid was withdrawn from the right pleural cavity (by means of an exploratory syringe). The temperature is slightly raised; pulse 90. The bowels are regular, and micturition normal. Urine s. gr. 1014; acid, contains no albumen and no sugar.

The *diagnosis* of this case was somewhat difficult at first—yet the onset with pain, the degenerative reactions, and the absence of the tendon reflexes noticed so soon after the onset, and affecting even those muscles which were not quite paralysed, made both Dr. Morgan and myself diagnose the case as one of peripheric neuritis rather than one of subacute poliomyelitis. Though the affection commenced with a good deal of pain and there was not much altered sensibility to be noticed after the patient's admission, yet there was a good deal of muscular hyperæsthesia as seen by pressing the calves or the muscles of the forearm. A symptom not usually seen in peripheric neuritis was the flexed condition of the knees—which, however, soon diminished after the patient's admission.

The *progress* of the case was very rapid and very favourable. The patient was treated with the galvanic-faradic current, according to Dr. Morgan's plan, and the patient soon commenced to improve; he was soon able to straighten his legs, and to move them. The atrophy of the muscles of the thumb and little finger gradually improved, and with these the other paralysed muscles. The tendon reflexes reappeared, and the patient left the hospital quite convalescent. The pleuritic effusion was also completely absorbed after a time.

Of the *third* group of atrophies, where neither cord nor peripheric nerves are affected, and where the affection seems to be a primary myotic, I would like to report one, where the symptoms were those of Erb's juvenile form, and where death took place from broncho-pneumonia. Unfortunately the notes of the case are very incomplete, as the patient was very ill at the time of admission, and died in a few days.

A. L., æt. 42, came to the Out-patients' Department of the Manchester Infirmary, Dec. 19, 1884, complaining chiefly of cough, and pain in the chest. On examining the patient, the marked contrast between the very atrophied arms and the still fairly muscular though somewhat emaciated forearms and shoulders; also the peculiar position of the scapula, and of the patient's inability to carry out certain movements, arrested our attention at once, and as the patient seemed altogether very ill, he was at once admitted into the Infirmary.

The patient told me that many years ago he noticed weakness of his right and then of his left arm without any pains. The weakness gradually increased, and was followed by weakness of his back and shoulder-blades. Eight years ago he was obliged to give up his employment as labourer, and was then employed as a checker in a mill; but latterly, partly from weakness in his legs and partly from the troublesome cough, he was obliged to give up this also.

On admission the patient, though somewhat emaciated, had evidently been a very muscular man. The cranial nerves and special sense organs were normal; of the muscles of the upper extremities, the biceps and triceps and supinator longus were very much atrophied; the muscles of the forearm and fingers appeared fairly normal, and the patient could execute any movements with these, whilst he could only raise his arms to the horizontal. He was unable to rotate them inwards owing to atrophy of the latissimi, or to adduct them owing to the paralysis of the pectoral muscles.

On the back there was marked projection of the inner border of the scapula, when the patient raised the arms and tried to carry them forward.

The deltoid muscle was impaired, and the patient was not able to hold his arm horizontally for more than a minute or two.

The patient raised himself with great difficulty from the recumbent posture.

The sensibility of the upper extremities appeared normal.

When walking the patient showed distinct lordosis.

The lower extremities showed the general emaciation, but the glutei seemed most affected ; the patient was able to adduct and abduct his legs well, and when sitting up, would cross one leg over the other, but when sitting had great difficulty in raising himself, and could only do so by the well-known manipulations. The calves were not markedly altered, they certainly showed no hypertrophy. Of the movements of the foot, dorsal flexion and adduction were well carried out, showing that the tibialis anticus was unaffected ; the peronei were, however, affected.

The knee-jerk could not be brought out.

The sensibilities of the lower extremities were normal.

The patient's general condition was very bad, and prevented a prolonged examination into the action of the muscles and also the determination of the electric reactions. He had a temperature of 103, pulse 110, and had marked dulness of right base, with numerous moist râles. The cough troubled the patient very much, and there was also profuse purulent expectoration.

The patient's condition became rapidly worse, and he died five days after admission.

The post-mortem examination showed extensive broncho-pneumonia on the right side, with dilatation of bronchi and some infiltration also of the left base.

The brain and spinal cord and peripheric nerves showed nothing abnormal in the fresh state ; the muscles appeared thin and pale, but to the naked eye showed no fat infiltration.

Microscopic examination of the spinal cord after hardening showed no noteworthy changes. The motor ganglia cells both in the cervical, dorsal and lumbar regions appeared mostly normal; some showed a good deal of pigment, others appeared smaller and at some places seemed shorn of their processes. Most of the cells, both in the cervical and lumbar region, however, appeared perfectly normal. In the white substance both in the lateral and posterior regions, a larger number of Deiter's cells were seen than normally. The anterior nerve roots showed no changes, and some of the peripheric nerves which had been kept for microscopic examination showed nothing abnormal. Portions of the pectoral muscle and of the biceps which had been put aside for microscopic examination showed no increase of the interstitial tissue, no fat-cells between the muscular fibres, but simply atrophy of the fibres, the striation in some places being not so well marked, and some slight increase of the muscle nuclei.

The intramuscular nerves showed nothing abnormal; there was no increase of the interstitial connective tissue, and no increase of nuclei in the sheath of the nerve fibres.

Though the case was only a very short time under observation, and though the examination was incomplete, yet the symptoms and appearances were so characteristic that I have no hesitation in calling the case one of Erb's juvenile atrophy. The only point in which it differed from the cases given by Erb is the weakened condition of the deltoid; but though this was not acting as well as a normal muscle, yet it was by no means so atrophied as some other muscles; otherwise, both as regards the onset and duration, the distribution of the atrophies, the absence of fibrillar twitchings, and the absence of any sensory troubles, the case corresponded closely with those given by Erb.

Microscopic examination of the cord and peripheric nerves showed scarcely anything pathological. Though a few of the motor ganglia cells appeared smaller and the processes shrivelled, yet I noticed no vacuolation, no marked atrophy and hyaline appearance such as we see in poliomyelitis and progressive muscular atrophy; the increase of Deiter's cells again is scarcely of any pathological significance. The result of this post-mortem thus bears out Erb's view that the juvenile form, which includes all cases of pseudo-hypertrophic paralysis, is not due to changes in the cord or peripheric nerves. These purely negative results give us, as yet, no clue as to the exact pathology of this singular affection and the initial causes which lead to the muscular atrophy. The appearance of the muscles would lead one to think that the atrophy is secondary, and if so we have to fall back upon the terminal nerve plates as the starting-point.

(*To be concluded.*)

SOME POINTS IN THE PHYSIOLOGY OF ATTENTION, BELIEF, AND WILL.

BY JAMES CAPPIE, M.D.

"GIVE me a fulcrum," cried the ancient sage, "give me a fulcrum, and I shall move the world." "Grant me a few postulates," says the modern reasoner, "and I shall read you the riddle of the universe." An unchallengeable postulate, however, is almost as difficult to find as a stable extra-terrestrial fulcrum. The scientific "spirit of the age" walks by sight and not by faith. It revels in facts. It numbers and weighs and measures, it catalogues and describes, it compares and classifies. To make progress among the secrets of nature its highway is Experiment, and its watchword is Demonstration. For any interpretation of a natural phenomenon it demands proofs that can appeal to the senses, and it looks with wholesome suspicion, if not contempt, on mere "arm-chair" speculation.

The marvellous success in advancing knowledge, and in gaining power over the forces of nature that has resulted from its use, is convincing evidence that the scientific method of interrogation is sound, and that it should always be adopted wherever possible. But it is not always possible to apply the method. The nearer we approach the region of subjective phenomena, the more difficult it becomes to test particular interpretations by an appeal to experiment. The galvanometer may reveal agitation in a sensory surface, but it tells nothing about sensation. The convolutions of a dog's brain may be tampered with, but he will not describe to us his feelings. Consciousness alone can discriminate the facts of consciousness; and the character, or succession, or relation of these can only be described in terms of metaphysic. Theories of physical relationship here must at first be tentative, and at the best they will require to be stated in very general terms.

The argument must consist in the application of general principles; and, in choosing these, analogy balanced by common-sense must be our guide. In drawing our conclusions, we may be satisfied if these can be held with some moderate degree of probability.

In attempting to gain a closer view of the somatic relations of mind, my subsequent argument will rest on the assumed correctness of three postulates. The first of these is—That every manifestation of mind is correlated to a definite mode and sphere of brain activity. This may be emphatically insisted on, whatever be the view taken as to the nature of mind itself. For, to take the illustration so frequently made use of,—that of the relation of a musician to his instrument,—the volume and quality and harmony of musical sounds are immediately correlated not to the fingers of the player, but to the tremors within the instrument. So the outcome of mental action, even as revealed to one's own consciousness, is not simply the result of some ideal, self-acting energy asserting itself, but it depends also on the compass and quality and adjustment of a material organization. It, of course, follows, that if we approach the subject from the physiological side, it is simply impossible to avoid the phraseology of materialism, and therefore, for doing so, I shall make no further apology.

My next postulate is—That the activity of the brain is conditioned by the activity of its circulation. The blood is to the grey matter of the convolutions what atmospheric air is to burning fuel; it is at once a necessity and a stimulus. However favourable may be the arrangement of cell and fibre, the consciousness will fail to respond to any impression, and every cerebral function will be impaired or suspended, if the circulation be lowered below a certain amount.

It follows that, so far as physiological means are to enable us to understand how mind and brain mutually act on one another, a consideration of the laws that affect the distribution of blood, and the influence of local surroundings in modifying these, must be of the first importance. Yet this is what is very seldom attended to. Volumes have been written on the relationship of mind and brain with scarcely a single reference to this aspect of the subject. While considerable progress has been made in defining the immediate sphere of activity, in certain mental acts, and especially in mapping the centres for voluntary motion, very little attention has been given to the influence which the many peculiarities of the encephalic circulation must have on the mode in which the brain may exercise its functions.

The principal general fact in regard to the local distribution of blood on which I have at present to lay stress is that, as a rule, the supply to every tissue and organ is in proportion to the demand for it. When function is quiescent, the need is slight in comparison with that of active exercise, and accordingly we find that the circulation of any organ contrasts remarkably in the two states.

Considerable difference of opinion has existed as to how this is immediately accomplished. By many writers the vasomotor nerves seem to be regarded as veritable physiological demons, whose unsleeping vigilance foresees and provides for all local wants; and it is supposed that, while the whole motor force acting on the blood is supplied by the heart's action, these nerves so regulate the calibre of the smaller arteries, that they turn on or shut off the blood current as may seem to be necessary. While not disposed to question their great importance, a good deal might be said in favour of the notion, that the molecular agitation in the tissue itself has a direct influence not only in assisting movement by lessening friction, but by exerting positive energy in urging the current onwards.[1]

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[1] In recent works on physiology it has been the tendency to ignore, if not altogether to deny, the active influence of local molecular change on the capillary circulation. In the higher animals, however, we have what I would consider crucial evidence in favour of its existence. I allude to the portal circulation. Here we have a large mass of blood returned from the chylo-poietic viscera, which, before it can reach the heart, has to traverse the ramifications of the portal system of vessels. It will be at once admitted that considerable force must be required for the purpose. Now, if a *vis a tergo* alone be employed in moving the blood onwards, the whole stress so occasioned must in this instance be borne by the mesenteric veins. The backward pressure within these vessels will be as great as what is required to transmit the blood onwards to the vena cava. Is it in the least probable that the thin walls of these veins could bear such a strain? I rather think we have here evidence that, while the general circulation may be sustained by the action of the heart, certain forces acting at the capillaries give indispensable aid in transmitting the blood through the latter.

Into the controversy on these points, however, I cannot enter here. For our present purpose it will be sufficient if the intimate relations between demand and supply be admitted as a matter of fact; if we can assume that functional activity involves a fuller volume and more rapid movement of blood in the capillaries of a part than does functional rest.

The last postulate I have to submit is the one on which my subsequent argument must mainly rest; but, unfortunately, it is the one whose soundness is most likely to be questioned. It is—That the mass of blood within the cranial cavity can be neither increased nor diminished directly, nor, indeed, to an appreciable extent within short periods of time.

A general statement of the argument in its support may lie in a nutshell. The available cubic space within the skull being a fixed quantity, the bulk of its contents must also continue uniform. These contents being the brain-tissue, the blood and the cerebro-spinal fluid, no one of these can be altered without an inverse change in one or both of the other contents. Thus, if a degenerative nutrition cause wasting of the brain-tissue, we must have an increase in one or both of the fluid contents, and thus evidence will be got of extreme congestion, or of serous effusion, or both. For any such change, however, time is required. Again, no amount of general depletion can reduce the intra-cranial circulation until time is afforded to allow effusion of serum to occur, because no mechanism exists for immediately raising any fluid from the spinal canal. Neither, on the other hand, can any increased force of the heart's action make the intra-cranial vessels fuller, for the cerebro-spinal fluid cannot be immediately displaced. It cannot be pressed into the spinal canal, for the latter cavity is already tensely full.

Referring to the classical essay of Dr. Kelly[1] for experimental proof of the difficulty of affecting the mass of intra-cranial blood, I content myself here with a single argument, looking at the subject from a common-sense point of view.

Whatever opinion may be held as to the nature of nervous energy, the phraseology used when its discharge is spoken of implies a certain amount of stress in the nerve-centre. Thus,

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[1] 'Transactions of the Medico-Chirurgical Society of Edinburgh,' vol. i.

"vibration" cannot occur without *tension*, and "explosion" implies previous *repression*. A chord will not give a clear tone when it is relaxed; and if the chamber in which a cartridge is exploded is not perfectly rigid, the effect on the bullet is weakened. If we are at liberty to reason from analogies like these, we must infer that no nerve-centre can have its energy economically liberated unless its structure is subjected to a certain amount of stress. Now, stress in a nerve-centre means stress in its circulation, and this involves pressure outwards and equally in every direction. If the energy is to be liberated with ease, and with exactness as to amount and direction, support to the structures immediately concerned must be as little yielding as possible. But if the cerebro-spinal fluid is at liberty to flow and ebb as readily as some writers assert, this steady support would be absent. The brain in such a case would resemble an instrument with slackened strings, and would refuse to give a clear response to impressions. Sudden, or powerful, or exact voluntary effort would then be simply impossible. For here, as everywhere, the discharge of pent-up energy will take place in the direction of least resistance. If the displacement of the organ's support occur more readily than the production of the intended result, such as the movement of a limb, the latter will not be successfully accomplished. Some of the energy would be wasted in the form of simple mechanical effect on the surroundings, and the result, whether mental or motor, will be less precise than would otherwise be the case.

The inference, then, is obvious. If time be an essential factor in the production of any change in the bulk of the brain-tissue, or in that of the cerebro-spinal fluid, then for the time being the mass of intra-cranial blood must also remain a stable quantity.

If we are allowed to assume the correctness of our last postulate, two corollaries require to be kept in view in applying it to encephalic physiology. In the first place, no change can take place in the circulation of one portion of the brain without that of some other part being inversely affected. In the abdomen, a determination of blood to one organ need not of necessity involve a diminished supply to the rest of the cavity, but an analogous occurrence within the skull is impossible. If the anterior cerebral arteries, for example, have their supply augmented, then to an exactly corresponding extent a lessened amount can be present in the other encephalic vessels.

In the second place, no change can occur in its circulation without a change in the balance of active pressure through the brain. The stress through the whole cranial cavity must, of course, be equalised, from the amount of fluids present, but the displacement of solid particles must occur, and such displacement is not likely to be without physiological significance.

Assuming the approximate soundness of these principles, we have to consider how they may be applied in encephalic physiology. My immediate object will be to show, that they must be of essential importance in any study of the correlations of mind and brain.

The first subjective condition or faculty I have to notice on its somatic side is Attention. It is unnecessary to enlarge on the psychological importance of this function. It may be said to underlie every other mental faculty. It is the bringing of the consciousness to a focus in some special direction. It is required to convert sensation into that comprehensive grasp of particulars which constitutes perception; without it, meaningless reverie will take the place of coherent thought; nor can we conceive of any act being strictly voluntary apart from its guidance.

To study it in its physiological relation, we may for convenience take the well-known effect of attention in modifying the intensity of sensation. The mental effect produced by an impression on a sensory surface is stronger, and details about the impressing cause are more completely gathered in when the mind is concentrated on it. On the other hand, if the consciousness is engrossed in some other direction—if absorbed by an interesting occupation or train of thought—the impression which formerly produced so much effect is felt obscurely or not at all. To account for this difference we cannot be content with a merely metaphysical explanation. To say that the mind is so constituted that it cannot at one and the same moment entertain with equal distinctness dissociated ideas, is only one half of the truth. There must be a cerebral correlative, and some notion as to the nature of this must be got if we are to come nearer the whole truth.

Two factors, at least, may be specified as bearing on this problem. In the first place, when the consciousness is engrossed by an immediate sensation, the sphere of encephalic activity is comparatively restricted. What that sphere may be in any particular instance it is for anatomy and experiment to determine. For receiving the impression, for quickening the consciousness, and for completing its course as a definite perception, the track involved may be wide and branching, but it does not include the whole brain.

In the second place, *the encephalic circulation will be focussed in the direction of activity.* The molecular agitation occasions a necessity and an attraction for more blood, and determination of this takes place all the more freely on account of the quiescence of the larger part of the brain. The latter has, as it were, loosened its hold on the circulation, and the impetus toward those parts which have an attraction for it is thus all the stronger. The increased activity of the circulation then reacts on the energies of the tissue, and the mental effect produced is therefore greater.

If now we turn the picture, we find the lights and shadows have changed places. Let the mind be intent on solving some problem, or be engaged on some work requiring close attention and nicety of handling, and the impression which formerly so completely took possession of the consciousness may now not in the least be felt. Here, too, physiological conditions are at work. The impression fails, not simply because the consciousness is otherwise engaged, but because the track along which it is to travel is not now in a fit condition for responding to the stimulus. It is out of focus. The momentum of the circulation is now directed toward the centres of ideation and voluntary motion, and this implies a derivation from, and consequent weakening of, functional vigour in the sensory ganglia.

If the above reasoning be legitimate—even approximately so—it becomes a matter of detail to apply the principle in other directions. In speculating on any point in mental physiology, we have something more than the molecular action of the brain itself to consider. The capillary circulation, too, has its laws, and the encephalic circulation its peculiarities, and a certain balance in the latter must be maintained if cerebration is to be healthy and its outcome exact.

In perfectly normal action it is likely that the molecular changes are the dominant factor and keep the circulation under control; but not unfrequently the mass and velocity of the circulating fluid may determine the sphere as well as the character of the activity, and thus have effect on the outcome, whether muscular or mental.

If the cells of one centre or class of centres be too readily explosive, they may attract the blood so strongly as to inhibit the function of other parts of the brain by the comparatively anæmic condition these are thus left in. Of this we have an illustration in the phenomena of an epileptic seizure. Here we have the blood determined in such volume to the motor centres that those which are more immediately related to consciousness have not sufficient left to enable them to sustain function with. Some writers seem to insist that during the seizure the whole brain becomes almost bloodless; but it would be as philosophical to expect a water-wheel to revolve violently by its supply of water being cut off, as that the energy of the brain can be prodigally expended in defiance of ordinary physiological conditions.

On the other hand, if the attractive power of some centre is under the normal, this may allow a determination to other centres to be excessive, and in this way, again, the action of the whole encephalon may be modified.

The means usually adopted to induce the hypnotic state afford us an illustration of a mode by which this condition may be brought about. In the first place, the attention is so strained in one direction that fatigue of a motor and of a sensory centre has been induced. When this has happened, the molecular agitation that accompanies activity of function becomes more difficult. Repose is needed to restore its former fitness for work. The structures immediately involved are reduced to a condition approaching that of sleep, and as a result they have relaxed their hold on the circulation. The forces which sustain its balance are therefore disturbed. The condition of the encephalic circulation may now be considered analogous to that of the atmosphere with a low barometric pressure; it is mobile and disposed to storms. If attracted in one direction, it is determined strongly. Then, the very momentum with which the blood surges in that special direction, reacts on and strengthens function. If it be toward an ideational centre, some particular idea may so monopolise the consciousness, that the judging faculty is almost as completely in abeyance as in ordinary dreaming. Thus, when something bitter is put into the mouth of a hypnotised subject, and he is told it is sweet, the notion of sweetness becomes dominant because the circulation is so strongly focussed toward an ideational centre that the gustatory centre or track cannot respond to its natural stimulus. Its function is suspended on account of the failure of a necessary condition.

In regard to volition, we need not here enlarge on the metaphysical subtilties its discussion has given rise to. We have now to do with it as a faculty subject to the tyranny of organic conditions, and our endeavour is, if possible, to catch at least a glimpse of its mechanism.

The Will is essentially a prospective faculty. It must have a goal in view, whether a muscular movement, or an effort of memory, or a process of reasoning be required to reach it. Some notion must precede action, and cerebration does its work without revealing to the consciousness anything of the mechanism employed.

Certain of the factors, however, may be specified with some precision. I, of course, must assume we have to do with an educated brain, in which co-ordination has been established among its various parts.

Ideation, then, the initiatory stage of volition, involves, within a limited area, molecular movement with corresponding vascular excitement. The function of that area becomes active, and *radiation of energy must take place in some direction.* Strands for the purpose of conduction branch off in innumerable lines to other centres. What the immediate direction may be, will of course depend on the circumstances of the moment and the results of previous association.

In order that the outcome may correspond with the intention, the impulse itself must be sufficient in intensity; the "points," (in railway phraseology) must be "open," the line must be clear, and the circulation must be so balanced that it is ready to surge in the required direction. The outcome may be defective by the failure of any of these conditions, and an improved adjustment in some needed respect may at once enable a correct result to be got. Thus, a familiar name or word may escape the memory, and for a time every effort may fail to recall it. Then, for example, a more successful focussing of the circulation taking place in the ideational centre, the word comes up without apparent effort. From this point of view, "unconscious cerebration" means simply, "better adjustment."

If the radiation be toward a motor centre, we find in the latter all the conditions favourable for liberating its energy. In the waking state, and in the absence of fatigue or disease, the centre requires little more than *permission* to do its work. All the potential conditions for discharge are already there. With the stimulus communicated from another centre, we have simultaneously molecular agitation and vascular excitement. The latter acts in two ways. In the first place, by derivation, it removes the inhibitory action of other parts of the brain; and in the second place, it further directly stimulates the molecular movement. The immediate result is turgescence or orgasm in the centre itself. Then, the general law in physics, that action and reaction are equal and contrary, must here hold good. If the surroundings, therefore, be stable, natural relief will be got by the overflow or discharge of energy into a motor nerve, and contraction of a muscle will be the result. On the other hand, if the support afforded to the centre be insufficient, the vascular turgescence will to some extent spend itself in displacing the surrounding tissues, and the intended movement will either not take place, or it will fail in precision and strength.

My object in the present paper has been rather to give prominence to what I consider a neglected factor in cerebral physiology, than to attempt its application to all possible instances. I shall be satisfied if I have said enough to show the importance of investigation in the direction I have indicated. Indeed, when one reflects on how much research is devoted to the minute structure of the brain itself, it seems surprising that so little attention is given to what may be called intra-cranial physics. We have here a field which is at least as likely to be fruitful in results as the attempt to measure and classify all the varieties of nerve-cell, or to unravel the complex network of nerve-fibres.

Within the skull we have an *imperium in imperio,* where, with loyal fealty to the interests and claims of Imperial unity, the rights of "Home Rule" are jealously conserved. As the speck of protoplasm requires the restraint of the cell-wall to enable it to develop and exercise some specific form of energy, so the brain-mass has its form moulded, and its development directed, and, especially, its energies exercised under the severe repression of membrane and bone. Then, in the approximately —if not for a time absolutely—stable mass of blood in the encephalic vessels as a whole; in the arrangement of the vessels themselves; and in the relation of their contents to the pressure of the atmosphere, we have conditions which contrast so remarkably with those we find in any other part of the body, that a consideration of their significance is surely deserving more attention from physiologists than it has yet received.

ON FALLING.

BY SAMUEL WILKS, M.D., LL.D., F.R.S.

Amongst the various symptoms which a medical man is called upon to interpret, there is none more important than that of sudden falling. He usually endeavours to distinguish between an epilepsy and syncope, although these two conditions by no means cover all the possible causes for the occurrence; if it has taken place without loss of consciousness, he has to seek much further for its antecedents. He has therefore to discuss with himself some of the most difficult problems connected with the working of the nervous system, although fully aware that no perfect interpretation can be made of his case until these have been solved by the physiologist. Nevertheless he makes use of the knowledge which he possesses, to guide him as far as possible towards the light. One of the most important facts which the clinical observer has of late gained from a study of disease has been independently worked out in the laboratory, viz. the much more intimate connection between the nerve-centres, nerve-fibres and muscles, than was ever before conceived. A nerve now is looked upon as the actual process of a cell, and its termination in end plates as closely incorporated with the muscle. No longer is a grey centre regarded as a mere store-house of electricity; the nerve a telegraphic wire; and the muscle a receptacle of force, like a packet of nitro-glycerine, ready to be exploded by the pull of a string, but the very nutrition of the muscle and its capability of storing up energy is known to be dependent on the nerves to which it is attached. It is obvious, therefore, that all changes in the motor centres will be exhibited by some corresponding alteration in the muscles. This has become a truism, so that the diagnosis of a central lesion is made when no other indication of it exists than a weakened state of some set of muscles. We daily speak of the blocking of an artery in the brain, or the rupture of a cerebral vessel, when not a single characteristic symptom of disease of the organ is present; we judge only by the fact of certain muscles associated through the nerves and spinal cord with a particular part of the brain being paralysed. This close union of nerve activity and muscle movement is recognised through the whole animal kingdom. The position and attitude of an animal testify to the working of the brain within; whether it holds itself ready to pounce on its prey, or lie crouching with fear.

In man the attitude of defiance, with well-knit muscles, and that of the trembling coward, exhibits at once the close connection between the muscles and the nervous system. The different positions of the hands and limbs as indicative of the ever-varying passions of the mind have been fully illustrated by Dr. Warner in his work on "Physical Expressions." The hard physiognomy of Julius Cæsar and the more supple features of Homer are seen in striking contrast in their respective busts in the British Museum.

The close correspondence between the nerve-centres and muscles is well exemplified in the exhibition of the so-called "thought reading." Not only does the nerve-centre stimulate the muscle to action, but the mere thought of a movement will influence it, as if the motor region of the brain could not in mere imagination be employed (if we may use so roundabout an expression) without a corresponding effect on those muscles which would be engaged in the movement; so that if the subject of the experiment be told to fix his attention closely upon the thing which he has hidden, the muscles which would have taken him to the place of concealment undergo a tension which is quite capable of being appreciated by any one who chooses to cultivate by practice this manipulative art. It is the unconsciousness of this change in his muscles which makes the subject of the experiment believe that the operation is a purely mental one. I apprehend that this is a law of our nature, and that in various processes of thought the different muscles which would outwardly express them are put into action, so that some persons with little power of restraint think aloud. I have been more than once struck with this when driving along the streets on the occasion of some damage to one's carriage, or of one's horse having gone lame; the common or uneducated people will openly proclaim the fact. It no doubt has also passed through the brain of every one on the road, but the better educated inhibited the outward expression of it. I mention in my published Lectures the case of a singer, who, when reading a piece of music mentally, "hums" the air; but if he has a sore throat, or is hoarse, he cannot appreciate its merits in the same degree as when he is well.

Mr. Francis Galton[1] made some very interesting observations, according to his usual scientific and rigorous method, by which he measured the boredom of an audience by the tone of their muscles. If the discourse was dry, the persons lolled and held the most varying inclinations towards one another; but as soon as the speaker said anything interesting, the audience braced themselves up, presented a fixed attitude, so as to become equidistant from one another. Mr. Galton thinks by a little trouble he could frame an exact measurement of the intentness or indifference of an audience.

It is evident, then, how intimately associated are the motor regions of the brain and the muscles; when the latter are braced up, the former are active, and there is a corresponding condition between the laxity of the one and the inactivity of the other. Not only is the connection of the nerve-centres in the spinal cord necessary for the nutrition of the muscles and accumulation of energy, but it seems as if the higher centres must be in action for the continued preservation of their tension. Let the activity of the former for a moment cease, as in sleep; the muscles become relaxed, the head drops on one side, the mouth opens, and if the individual be standing, he falls to the ground. There seems to be a constant need of action in the higher motor cells to preserve the body in position, for between contraction of the muscles on the one hand, and complete relaxation on the other, there is an intermediate condition of tension necessary for this preservation of the balance of the body; there must therefore be a state of nerve centres both of cord and brain, which is one neither of great energy nor of repose, but one of continuous action.

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[1] 'Nature,' June 25, 1885.

If we have a patient who is subject to falling, it is evident that the higher motor regions, or some part of the lower track leading to the muscles, has for the moment become inert, and we ask ourselves in what way has this been brought about. One of the best known facts in reference to the physiology of the brain is, that its function is dependent directly upon a due supply of blood; not only is the latter necessary for its nutrition, but for its momentary activity. If the circulation be arrested, unconsciousness occurs instantly, and the person falls. It is evident that a general cessation of the circulation would result not only in unconsciousness, but in a loss of other functions of the organ; it does not, however, follow that there might not be an arrest to the flow of blood in some portion of the brain, leading to a paralysis of certain muscles without loss of consciousness, and no doubt this does often happen in hysterical women. The sudden falling in syncope appears clearly to arise from an arrest of the flow of blood to the brain, as the patient becomes pale and pulseless, and the local effects of arrest are seen in the case of hemiplegia, resulting from ligature of the carotid. As it is important to bear these great facts in mind, it may be as well to refer to the experiments of Sir A. Cooper, performed more than fifty years ago.[1] He commenced by tying the cerebral vessels in dogs, and observing the effects. He first ligatured the carotids, which produced some temporary disturbance in the respiration and circulation. As soon as the animal was restored, he exposed the vertebral arteries and compressed them; the dog immediately became unconscious and seemed to be dead; he removed the pressure, and the animal as speedily recovered. He compressed the vessels a second time, and then, as before, there followed loss of motion and apparent death; when the ligature was loosened, the animal came to, with a deep inspiration. After some hours he compressed the vessels a third time, and repeated it in all six times, and always with exactly the same result. Sir Astley observed that this compression of the vertebrals after the carotids were tied put an entire stop to the functions of the brain. He then took another dog, and tied the vertebrals; this produced a greater effect, as it seemed to paralyse the forelegs. When on the following day the animal had recovered, he compressed the carotids, immediately it fell, losing all sensation and volition; on removing the pressure it soon recovered. He performed this experiment four times with the same result. He then took another animal, and tied all the four vessels; it ceased to breathe, and soon died. Sir A. Cooper adds, "this is a most decisive experiment, showing the effect of the arrest of blood in the vessels of the brain in stopping respiration, volition and sensation, the result being striking and immediate." Sir Astley then tried the effects of pressure on the neck, with the same results, or in his own words: "The same effect of interrupting the stream in the vertebrals and carotids was produced in an equally conspicuous manner without the application of ligatures, as follows: The animal was held in a convenient position, with its neck extended and its head thrown back. I then applied my thumbs so as to compress at the same time the two vessels on each side, taking care to leave the trachea entirely free from compression. Respiration ceased in a few seconds; some struggling then took place, and the animal appeared dead. The pressure being then removed, the respiration was completely suspended; but artificial motion being given to the ribs, the animal gasped, began to breathe quickly and recovered. I also put a ligature around the neck, close to the sternum, so as to compress the carotid and vertebral arteries, but the trachea was excluded by passing the ligature behind it. Although the trachea remained free, as soon as the ligature compressed the carotid and vertebral arteries, breathing ceased, and all the functions of the brain were destroyed."

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[1] 'Guy's Hospital Reports,' vol. i. 1836.

Not being quite sure that he might not have injured the nerves, he proceeded to put ligatures on the pneumogastric, phrenic and sympathetic nerves, or in his own words: "Before I would venture to draw conclusions from the experiments above detailed, I was desirous of convincing myself that no injury done to the nerves could have influenced in any material degree effects which had been observed. I proceeded therefore to investigate the consequences of applying ligatures to the principal nerves of the neck."

Sir A. gives the results of these experiments, which were negative as regards the object he had in view, and concludes his paper with these words: "The sudden death that takes place from pressure at the sides of the neck must not be attributed to an injury to the nerves, but it is owing to the impediment to the due supply of blood to the grand centre of nervous influence."

With our improved experimental methods I do not know that physiologists would be altogether satisfied with Sir A. Cooper's conclusions, and more especially as it is now generally believed that an inhibitory action can be produced on the different organs of the body through the nervous system, and that the brain can be included in the general law; indeed it is probable that cerebrum, cerebellum and spinal cord may possess some inhibitory power over one another. It may be a question, therefore, whether some direct influence may not be exerted through the nerves, either on the heart or the brain, in such an experiment as that made by Sir A. Cooper by pressure on the neck. Dr. de Watteville informs me that he has often performed the same experiment on the human subject with like results: by placing his thumbs on each side of the neck, and exerting a little pressure, he can make the strongest man fall at his feet. He believes that he compresses the vagus, and that it is through this nerve that the effect is produced. Czermak, it may be remembered, was in the habit of showing how by pressure on this nerve he could influence the action of his own heart. These opinions, it is seen, are in favour of the brain being influenced through the heart.

I have long known, and been in the habit of teaching as an important practical fact, that pressure on the neck, as in hanging or garotting, will often produce instantaneous insensibility and death; and that strangulation by no means implies congestion of the lungs and engorgement of the right side of the heart, as writers on medical jurisprudence have maintained. The fact is of the same nature as that exhibited in Sir A. Cooper's experiment. Even death by obstruction of the larynx is sometimes instantaneous, and not accompanied by the usual symptoms of suffocation, as if there had occurred a sudden paralysis of the respiratory centre.

That the brain can be acted on directly independent of the heart would seem to be proved by cases of shock, whether this be physical or moral. A blow on the head of a man at once "makes havoc among those tender cells, and checks his power to shape," and at the same time produces a paralysis of motion and of all the senses. A fright will produce exactly the same result, as I have witnessed on several occasions in the cases of women who have lain for days in a state of torpor or trance after some dreadful catastrophe. When under similar conditions the patient has been paralysed on one side only and retained her consciousness, we cannot but believe that one half of the brain has been inhibited in action, as in hysterical hemianæsthesia. If the same result should follow an injury to the head, as I have on several occasions seen, it would follow that a blow might be sometimes given with that exact amount of force which would concuss one hemisphere of the brain and not the other. All these cases show that the brain can receive a direct influence through the nervous system without introducing the instrumentality of the heart's action. Nevertheless the functionising power of the brain does seem to be momentarily dependent upon the heart. If this organ stops, the individual is dead; a blow on the abdomen has been known to arrest suddenly the heart's action, and death is instantaneous. Were it not so, we should have to believe the ghastly French stories of guillotined heads talking and grimacing. In favour of direct inhibition of the brain through nervous influence, I might mention the case of wild animals suddenly expiring from fright, and the lesser effects produced upon them by the so-called mesmeric process, whereby they will lie for a long time in a completely torpid state. A sudden strange noise will cause my parrot to drop from its perch as if it had been shot; an inhibitory action on the brain through the auditory nerve. I have purposely avoided speculating upon the supposed inhibitory action of the brain on the cerebellum or the spinal cord, as the question has not been satisfactorily determined, being content rather to speak of the clinical facts which one is anxious to elucidate. These seem rather to show that the activity of the cord is not only associated with, but dependent upon that of the brain.

All the instances mentioned show how directly the healthy working of the muscles is under the influence of the brain, and that the activites of the two structures correspond. In sleep, the brain is inactive and the muscle relaxes; in close attention, the muscle contracts; and in a determined state of mind, as in anger, the muscles would be tightly knit up. Just as the passions of the mind vary in kind and degree, so I apprehend the state of the muscles varies, and similar degrees of contraction occur under simple mechanical conditions. If, for example, the arm be bent by flexing the muscles of the elbow, it may be retained in that position for a long time without fatigue; but if any weight be placed in the hand, the flexors have to exert a greater power to overcome the strain, and I apprehend the contraction of the muscle in the two cases means two different conditions; there is evidently a greater expenditure of force in the latter case, with a corresponding fatigue; but what the difference is in the anatomical constituents of the muscle I would not venture to say. Since the natural tension of the muscle is only preserved whilst it is attached to a healthy nerve-centre, it follows that its integrity and reinforcement after exertion must depend on nerve influence; but since during the stimulation of the motor nerve it is undergoing exhaustion, it necessarily follows that it is during its relaxation and state of rest the muscle is acquiring new vigour. The energy of the muscle is produced in itself, and is not a direct power sent by the nerve, for if not so, a horse would not be stronger than a man. The muscles are like accumulators in which a chemical force is stored up ready to exhibit itself as electricity, but which can only be produced under the influence of a battery. The subject of degeneration and repair of muscle has been admirably discussed by Dr. Gaskell in a paper in the 'Journal of Physiology.' He maintains that there is good reason to believe that all the muscles are like the heart, innervated by two sets of nerve fibres of an opposite character; and therefore, as motion tends to wear out the muscle, so there is an inhibitory nerve which stops all action, and at the same time allows the renovation of the muscle to take place.

All the questions involved in the subject which I have been discussing are by no means exhausted, for we have taken no heed of the intermediate apparatus between the motor brain-region and the muscles. Seeing that the fibres of the motor tract proceed mostly to the anterior cornua of the cord, and from these again other nerves pass on which stimulate the muscles, it is probable that nearly all the cerebral influence is conveyed through the spinal system. The size of the cord would be opposed to the idea of a more direct connection, and we know that there is no need of it, seeing that the muscles act in groups under the direction of special centres, as for example in the process of talking, where a large number of muscles innervated by numerous nerves are set in operation through a centre which is regulated by the brain. If this is true, the contrary is true; and when the brain is inert, the spinal centres are at rest. We have nothing to show (except speculatively) the inhibitory action of one on the other. Of course in disease a so-called cerebral and spinal paralysis are very different. Any lesion between the brain and spinal centre, whence proceed nerves to a limb, does not affect in any way the nutrition of the latter; the limb is healthy, but it cannot be reached, as the line of communication between it and the volitional region of the brain is severed. A lesion of a spinal centre, however, or of the nerves proceeding from it, produces loss of nutrition of the muscles and their speedy atrophy. I know nothing of such a result as this arising from a mere inhibition of the function of the cord, but an inhibitory arrest of function of certain parts of the cord must, I apprehend, continually occur. That the cord can be stopped in its ordinary physiological work without any structural change occurring in it, is seen in cases of angular curvature, where a patient may be totally paralysed for many weeks and then perfectly recover. To say that the cord has been pressed upon, does not explain how the pressure has caused it to cease work without any material damage having been inflicted upon it. In the acute ascending, or Landry's paralysis, no change may be found on the minutest examination of the cord.

That the cord may be the seat of an inhibitory influence I infer from the fact, that in some cases of very chronic spine disease, patients have suffered, amongst their earlier symptoms, from sudden falling. A young lady, now hopelessly paralysed, used about two years ago to suddenly fall whilst walking; she would then rise and proceed as before. On the closest investigation there did not seem the slightest suspicion of even a momentary loss of consciousness. And in the case of a man who died in the hospital of general degeneration of the cord, his first symptom was falling in the street. In other cases which I have not had an opportunity of following, the result has not been known. I mention in my published lectures, under the heading "transient paralysis," the case of a butcher who whilst carrying weights would often suddenly fall, at other times lose the power of speaking. Without any apparent reason seizures would come on, when his speech suddenly failed, and he was quite unable to move his limbs, swallow, cough or spit. There was no symptom of epilepsy, and no spasm. I also mention the case of a gentleman who would suddenly lose all power of walking, and sometimes would find he was unable to speak. This was not due to spasm, but to relaxation of the muscles, for his jaw would drop, and he was unable to close his mouth. His wife often held up his jaw until power had returned. To use his own expression, everything was going on regularly in his body, when suddenly there was a reversal of the engine. He eventually lost these attacks. I was in the habit of illustrating to students the difference between paralysis arising from organic disease of the cord, and that arising from a mere inhibited action, by taking two watches which were not going. It was impossible to say from looking at the exterior what was the cause of this; but on examining the inside, we found one seriously damaged in its chain or spring, whilst the other was a perfect watch, but had not been wound up. Many slighter forms of transient paralysis one occasionally meets with, as when a patient describes a sudden weakness coming over an arm, and any object which was being held falls to the ground. A gentleman for two years after typhoid fever, although he could walk well, would be occasionally and temporarily seized with almost complete paraplegia.

It will be seen that where falling has occurred with loss of consciousness, we have placed the cause in the brain proper; and when there has been no loss of consciousness, we have looked for a cause in the motor tracts below, or in the spinal centres; but there seems no reason to doubt the possibility of the function of certain motor areas being inhibited alone, just as these may be disturbed alone in cases of syphilitic disease accompanied by hemi-convulsion without loss of consciousness. As I have before said, I believe the hysterical hemianæsthesia is due to an inhibitory influence operating on one hemisphere, for there is not only a paralysis of half the body, but accompanying this there is often an obliquity of the mind. That consciousness remains when a large part of one hemisphere is destroyed has frequently been observed.

Since in sleep there is the same loss of consciousness and paralysis of the muscles as in syncope, I have assumed that the cause is the same in both; a diminished circulation in the brain. This has been proved to demonstration in the case of sleep, and is exactly what might have been imagined by considering the varying function of organs. Sleep, which has been regarded as one of the most mysterious phenomena of the animal economy, is not peculiar to the brain, but is merely the most striking example of the alternating action of the functions of the human body. We have just now seen how the muscular apparatus wears out, and is again replenished by rest; and as for the stomach, it is quite a different organ when in action and at rest. In the latter condition it is small, with its interior pale, containing little blood, and its secretion scarcely acid: the organ is asleep; but immediately food is put in it, it wakes up, blood flows into its structure, an acid secretion is poured out, and it begins to act in a peristaltic manner on the material within.

Readers may be disappointed at my not arriving at definite conclusions on the subject so important and interesting as that which I have been discussing. I am of opinion that the time is yet distant when the questions I have raised can be satisfactorily solved, seeing that they include some of the deepest problems in the physiology of the nervous system. I have merely in a desultory manner spoken of a symptom with which we are constantly meeting, and expressed some thoughts which necessarily arise in considering it.

A FEW NOTES ON HYSTERIA.

BY JAMES OLIVER, M.B. (EDIN.), M.R.C.P. (LOND.)

Hysteria, like epilepsy, is a functional disease of the nervous system, and since it may affect the whole or any part of the cerebro-spinal tract, the symptoms, irregular and variable, cannot be included under one rigid formula. An exhaustive description of the disorder would be tedious and unprofitable. I shall therefore merely attempt to note a few details regarding its etiology, and pass on to discuss a few of the more characteristic signs. The appellation "hysteria" is at best faulty, conveying, as it always does, misapprehension to the minds of outsiders, and employed, as it too frequently is, as a cloak for our own ignorance, or lack of observation whenever unable to otherwise relegate existing symptoms. Let us ever beware and guard against making this reprehensible error, for many unfortunate sufferers, who have evinced no other manifestations than those of a so-called hysterical type, have ultimately succumbed, with no special or marked alteration of symptoms, the victims of true organic disease. In many cases, if not all, the organic disease has existed from the onset, and is not, as many authors would have us believe, a mere sequel of the functional disturbance. Occasionally I have witnessed autopsies on the bodies of those who, during life, were the supposed victims of hysteria or hypochondriasis, and at which no trace of organic mischief was revealed. The simple fact, therefore, that hysteria, as such, has hitherto eluded all detection is no criterion, and cannot justify the conclusion usually tacitly implied, that it has no other than a mere fanciful existence in the mind of the unfortunate sufferer. That the mental state exercises a mighty influence over the well-being of the body, no one can dispute, and it may be occasionally the dominating, if not sole factor at work, in producing functional manifestations.

Regarding the essential or fundamental cause of nerve instability, nothing definite is known, and the whole question, still wrapt in obscurity, may yet, we hope, be evolved under the guidance of the scientific mind free from all bias. So far it appears to depend upon some alteration in the relative amounts of the chemical constituents of nerve tissue, and in their mode of union, whereby there results an augmentation of the elasticity or tension, and consequent increased proneness to molecular trepidation. Through the medium of our nervous systems, each organ and every ultimate tissue of the body is made aware of, and acts and re-acts in harmony with, those multifarious changes for ever going on, not only in the organism itself, but its environments. When, however, we remember that higher organisation and greater functional activity are concomitants of lessened structural stability, it is not at all astonishing that a tissue so highly organised and functionally active as that of the brain and spinal cord of man should so readily be deranged. Considering the manifold influences at work in the organism itself and its surroundings, it is, to my mind, more a subject of surprise that deviation from the apparent normal state is not more commonly displayed.

The nervous system of the female is evidently more prone to manifest functional change than that of the male. Yet hysteria is not exclusively a disease of the female sex, for I have frequently witnessed its evolution as a traumatic sequel in the apparently robust and energetic labourer. In the female it usually appears between the period of puberty and the cessation of sexual activity. It is not, however, confined to this age. I have occasionally noted its existence in children, and recently to a most marked extent in a girl of seven. In both sexes it is a most intractable disorder, but is possibly more amenable to treatment in the female than in the male.

In many cases derangement of the nervous system is supposed to result from functional disturbance or true organic disease of the genital organs, and often no other associated or causal disturbance can be elicited. Here the influence may be of a direct nature, or occur through the agency of the emotions, for, be it remembered, sexual disturbances often prey upon the mind, and may thus indirectly produce prejudicial effects on the whole animal economy. With the approach and appearance of each recurring monthly flow, the whole female frame may, in health, and apart from any resulting emotional state, participate in the change. In some cases of organic disease of the nervous system in the female, menstruation is, I have occasionally noted, anticipated by the development of symptoms, otherwise latent, or the aggravation of symptoms already in existence; a fact strongly presumptive of a close relationship, direct or indirect, between the sexual functions and the cerebrospinal state. In many cases of hysteria, anomalous sensations are complained of; as, however, the majority of such are vague and indescribable, it would be futile to attempt more than a description of those commonly experienced. Anæsthesia, or loss of sensibility, may affect the lower limbs when it is usually associated with a simple paresis or complete loss of power, and reaches the same level on both extremities. It may partake of the form of hemianæsthesia, in which the whole of one side of the body is more or less completely involved. The loss of sensibility then may or may not coexist with motor manifestations, hemiplegia, hemiparesis, or hemichoreiform movements being at the same time displayed. Under such circumstances, the head and special senses on the anæsthetic side may escape; occasionally, however, they likewise participate in the change. Nor is this to be wondered at when we recall the fact, that the special senses are but highly organised modifications of the tactual. When loss of sensation is confined to one side of the body, it will be noted that a line passing through the middle of the body longitudinally, separates in a most marked manner the healthy from the disordered half. In a few rare instances I have elicited the coexistence of hyperæsthetic patches on the anæsthetic half of the body, more especially in the region of the ovary and breast, and this apart from menstruation. There can be no doubt that the hemianæsthesia is central, and results from functional disturbance of the higher cortical centres in the brain; whilst the simultaneous existence of hyperæsthetic patches depends on a markedly augmented tension of the peripheral nerve-endings located in the region manifesting the change. Pain is, in reality, an aggravated pleasure. The finger placed in tepid water may convey to the cerebrum a sensation of pleasure, whilst placed in boiling water it forthwith excites a sensation of intense pain. These two states are essentially the result of like peripheral changes, the one being merely a more intense excitation than the other. Of the special sense-disorders co-existent with hemianæsthesia, that of the visual apparatus demands especial note. The change manifested is not a hemiopia, but crossed amblyopia. There is dimness of sight with the eye on the anæsthetic side, contraction of the field of vision, and loss of appreciation of colour. The opposite eye likewise manifests, although to a much less extent, a diminution of its field of vision.

Hyperæsthesia, apart from anæsthesia and occurring in scattered patches, is a common manifestation of functional disturbance of the nervous system. The merest touch produces a sensation of intense pain, whilst deep and continued pressure is readily borne, a fact strongly in favour of the change being one confined, as a rule, to the nerve-endings. This symptom is not, however, one peculiar to hysteria, for I have seen it associated with true organic disease, and recently to a most marked extent in a female, the subject of disseminated sclerosis of the cord, in whom during life the dress, whenever it impinged against the legs in walking, elicited great tenderness.

The motor manifestations of functional disturbance of the nervous system are most varied and full of interest. Occasionally we witness a peculiar phenomenon, the so-called paradoxical muscular contraction, the result of some change in the muscle state, whether of the nature of a contraction or relaxation is still conjectural, but which allows of the foot or any other part of the body being so posed passively that it will maintain this position for a more or less indefinite length of time. Under favourable circumstances it is avowed this muscle state may be induced by kneading those muscles antagonistic to the attainment volitionally of the new position. By friction and the application of heat to the group of muscle apparently the seat of change, I have brought about complete relaxation and the cessation of the phenomenon, and this doubtless through a distinct alteration in the relationship of the muscle and nerve molecular state.

During health many muscles of the body act in association, the resulting movements being purely automatic and in reality produced without the intervention of the will. Every one is familar with the fact, that the will often tends to prevent movements which, if otherwise left to the guidance of the automatic centre, would be performed with the utmost precision, the mere intervention of the influence of the higher nerve-centres determining failure. It is not therefore astonishing that functional disturbance of the cerebrum should occasionally result in a disassociation of movements at other times harmoniously combined. Recently I witnessed the manifestation of this phenomenon in a female. The eyes were constantly closed, and any attempt to open them only resulted in a frown or contraction of the corrugators; whilst any attempt to close them more firmly was equally ineffective, and induced merely a contraction of the frontalis muscle, resulting in corrugation of the forehead. Elevation of the eyelids is a movement associated with contraction of the frontalis muscle, as every one may verify for himself on gazing at an object placed at some height above him. Frowning, on the other hand, is commonly associated with a more or less distinct closure of the palpebral opening. In the case I have just mentioned there was therefore evident lack of harmony in movement, or rather, total reversion.

Paraplegia, complete or incomplete, is a frequent concomitant of functional disorder of the brain and spinal cord, and here it is impossible to say whether the change is really in the cord itself or the cerebrum. Anæsthesia, more or less pronounced, often coexists, and although incontinence of urine is said never to result, yet I believe it may occasionally occur. The legs are invariably rigid, and reveal to a varying extent the phenomena induced by sclerosis of the lateral columns. Ankle-clonus is, as a rule, readily developed, but assumes a peculiar yet withal characteristic constructive type whereby instead of the regular to-and-fro rhythmical movement of the foot, there are evinced occasionally marked interruptions. In such cases it is also a most inconstant physical sign; whereas when the result of true organic disease, it is most uniform and constant. Hemiplegia is sometimes noted, but one limb is invariably more affected than the other, and there is no affection of the face, a sign of great diagnostic value.

Clinical Cases.

CASE OF LOCOMOTOR ATAXY, WITH CHARCOT'S JOINT LESION.

BY J. WALLACE ANDERSON, M.D.,

Physician to the Glasgow Royal Infirmary.

THE patient whose case is detailed below was in the first instance admitted to one of the surgical wards of the Glasgow Royal Infirmary under the care of my colleague, Dr. Fleming, who showed the case from a surgical standpoint to the Glasg. Path. and Clin. Soc. in October last.* The patient subsequently passed over to my care. His case is noteworthy, not only as an example of Charcot's joint lesion, but as presenting other interesting, possibly analogous, phenomena of a comparatively exceptional kind.

W. K. æt. 45, gardener, admitted to ward xii., 30th Oct. 1885, complaining of a difficulty in walking from weakness of the hip joints. He ascribes his complaint to a stroke of lightning which occurred twelve years ago. He was engaged at his work, and had just got under the eaves of an outhouse, when he saw a vivid flash and immediately felt stunned, but did not lose consciousness. He walked half-a-dozen steps and lay down on some matting, and after a few minutes he put down his hands on his legs "to feel if they were there," as he says, "for they were quite benumbed." After lying for about half-an-hour he was able to walk about again, and found that the lightning had struck the ground about forty yards off, doing great damage.

He has no recollection of any immediate effects besides these just mentioned, but about six months afterwards he began to feel "little tingling pains in the thigh just under the skin." These pains were not at first continuous. They kept quite away as long as he was at his work, but when resting, or even on stopping to speak to a friend as he walked along the road, they would make themselves distinctly felt. They

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[1] See 'Glasg. Med. Journ.,' Jan. 1886.

gradually became more severe and deeper, and were worse, as they still are, in cold, or on a change of weather. One day, about two years after the pains had begun, while at dinner, he suddenly felt very strange and lost consciousness for about ten minutes. But he resumed his work on the same afternoon, and the attack has never recurred.

What he recognises as the next stage of his complaint began four years ago. A kind of 'weakness' in the left hip joint was the first thing observed; he had no pain in it, but on lifting any heavy weight it was that joint which failed him; and finally one day it gave way so as to convince him he had dislocated it. He would say the weakness was felt for about a year and a-half before dislocation occurred, and a sense of creaking for three months previously; and this last has been felt ever since.

Immediately after the joint went out, a swelling appeared for the first time in the groin, "round and raised up," he says, "like a large closed fist." From other remarks of the patient, I think the swelling must have suggested femoral aneurism to a surgeon who saw it: the tumour, however, gradually disappeared.

Meanwhile the pains had become of a much more severe character, darting down as far as the feet, and sometimes darting upwards into the abdomen. Often at night he had to rise out of bed and walk about for very agony, or lie down before the fire wrapped in a blanket: heat and exercise being the only things that gave him relief. But he had no sickness or vomiting, or special gastric pain.

On the most careful enquiry it cannot be made out that there was anything more nearly approaching the gastric crises. He speaks of a squeamishness or distaste for food that continued for a month or two in the early part of last year, but he always finishes his account of this symptom by saying that he is sure it had nothing to do with his present trouble; but the girdle sensation was very marked at this time.

About the same period certain laryngeal symptoms presented themselves, which were fortunately noted by my colleague, Mr. Macintyre, assist. surgeon to the hospital; he says, "the patient observed that his voice, which before this was a powerful one, became very weak, and as he had been considered a good singer previously, the fact forced itself upon his attention. On laryngoscopic examination, the appearance of the true vocal cords is very much what we see in paralysis or paresis of the thyro-aryteno-interni muscles, i.e. the middle of the cords do not touch each other during phonation, and an elliptic aperture is the result. There is no paralysis of sensation or of motion in the pharynx or larynx beyond the above. The patient rather looked upon the loss of voice as part of the general weakness from which he suffered, and we must keep that possibility in view. Further, such a condition as that above described is found sometimes in the normal state."

The next distinct period in his illness as he himself details it, began in June last, when the right hip-joint gave way. It "went out" one day while he was in Glasgow. Then for the first time he was fairly crippled, and had to call a cab. Previously he had felt some weakness and creaking in that joint, but hardly over such a long period as in the case of the other. He was now quite confined to bed, and continued so until admitted under Dr. Fleming's care.

Giddiness has not been by any means a continuous symptom, although for some months before and after the dislocation of the left hip, he had frequent attacks of vertigo with dimness of vision. For example, on walking along the road, he would suddenly require to stop and steady himself for a few minutes on the wall or fence. But this symptom gradually disappeared.

Since admission, we have found complete absence of knee-jerk, and inability to stand on a narrow base with the eyes closed; but any peculiarity of gait is difficult to separate from what is obviously due to the joint lesion. Tactile sensation is impaired in the left leg as compared with that of right, and in both it is less than in the arms; but not delayed or repeated. From careful thermal observations, I have no doubt patient is unduly sensitive to the cold test tube. The muscular sense or appreciation of weight appears to be normal; the pupils are normal to light, and I think to accommodation, but he says the right eye is weak, and after reading a little he closes it with his hand, and uses his left eye alone. He has also double vision if looking at an object a few yards off.

The case having attracted the attention of Dr. Buzzard, I am indebted to him for suggesting the following inquiries which I have made more recently.

It appears that he perspired profusely on the slightest exertion for a period of ten or twelve months, about two years after being struck by lightning. The sweatings were not localised, but were so pronounced that he can easily fix the period of their duration, and he remembers they disappeared as suddenly as they came. He has had no choking sensations; no distress of breathing or palpitation; has had no cardiac murmur or anything suggesting structural alteration in the heart, at least since he came under our care, but the heart's action is markedly irregular.

No perforating ulcer; no shedding of the nails. He states, however, that two years ago, three teeth decayed and loosened, so that he could pull them out himself, whereas up to that time they had all been in excellent condition. I had previously made inquiries regarding the following symptoms presumably analogous to these just mentioned.

On the instep and front of the ankle of the left foot there is a faint discoloration that reminds one of a scorbutic blush just disappearing, and it appears that three years ago he had an itchy eruption there, that from his description must have been of an erythematous character, followed by slight desquamation. It continued for six or seven months, leaving the mark above noted.

There is no sugar in the urine; no hemi-atrophy of the tongue.

After getting with some care the foregoing details from a patient who has been led to give a precise but by no means exaggerated account of his symptoms, my difficulty still is to say how much is accidental and how much essential. He certainly seems to present unusual variations in his general condition, as if that were exceptionally unstable—to a greater degree than can be readily explained simply by his weak state of health. For a time he is bright and cheerful; he feels well. Then he is out of sorts; some pains, or a diarrhœa, or loss of appetite disturbs him, and he looks ill. I may say, however, that there has been a decided improvement in the patient's walking powers during the last six months. When admitted to hospital in October last, he could not walk a step unless firmly supported on each side; now he can walk downstairs and out of doors a little with the aid of his two sticks.

Dismissed, June 14, 1886, much improved.

CASE OF REMARKABLE HYPER-EXCITABILITY OF ALL THE MUSCLES AND TENDONS OF THE BODY TO DIRECT MECHANICAL PERCUSSION, WITH CLONUS OF VARIOUS REGIONS, INCLUDING THE LOWER JAW. RECOVERY.

BY A. HUGHES BENNETT, M.D., F.R.C.P.

A CASE of Clonus of the Lower Jaw, published in a recent number of 'BRAIN,' by Dr. Beevor, induces me to put on record the following case which has come under my observation, and which was kindly sent me by Dr. Francis Hawkins. The etiology and pathology of the condition is obscure. The patient will doubtless be considered as having suffered from hysteria, as this convenient neurosis has to answer for a multitude of mysterious phenomena, and to provide a refuge for our ignorance. This would be, however, at best, only an imperfect explanation of the condition about to be described. Physically, intellectually, and morally, this woman presented none of the protean or emotional characters which are usually associated with that disorder, and, with the exception of the symptoms from which she suffered, was in all respects of sound bodily and mental health.

Ellen C——, aged 26; single, housewife. The chief complaint of the patient is weakness of her legs, and the occurrence of frequent attacks of spasms in her extremities. The family history is unimportant. The patient herself has always been in good health till three years ago, when she had a discharge from the right ear, accompanied with deafness, which has continued since. Two years ago, without apparent cause, she was somewhat suddenly attacked with headache, vomiting, and general malaise. This illness lasted about a week. Ever since, she has been subject to seizures of trembling and spasm of the limbs, which occur spontaneously and without loss of consciousness. Her powers of walking, also, have become impaired, and her legs are weaker than formerly, otherwise her health has been good.

On examination, the general health is robust, and all the organs and functions normal in every particular, except the following exceptions regarding the nervous system. The intelligence of the patient is perfect; she is of a calm and stable temperament; there is no evidence of special nervousness, or signs of a hysterical character. In short, the patient has an unusually composed and well-balanced disposition. The special senses are all normal except the old-standing deafness of the right ear. Vision, and the appearance of the fundus oculi normal. There is no paralysis, tremor or nystagmus of the eyeballs. There is nothing abnormal to be detected in the condition of the face or limbs, the movements of which are normal, although the patient states that her extremities feel weak, and that she is unable to walk above a few yards without fatigue, and bringing on the attacks about to be described. There is no rigidity of the muscles or incoördination of their movements. Several times a day the patient is seized with a sort of cramp, sometimes in the left arm, sometimes in the right leg, and sometimes in both together. These occur either spontaneously, or as the result of a sudden movement of, or blow on the limb in question. This consists of a tonic spasm of all the muscles of the limbs which renders them rigid and distorted, and which continues for a few minutes, after which it quickly relaxes. In the upper extremity the arm is firmly adducted to the side, the forearm is flexed, the palm of the hand extends upwards over the shoulder with the fingers clenched. The lower extremity is in a state of rigid extension, the knee adducted, the foot in a condition of equino-varus. These are the most common forms of distortion, but they vary somewhat on different occasions. Immediately after these attacks the parts implicated feel weak, but soon regain their normal condition, and in the intervals the patient feels quite well in every respect. Besides these, the chief forms of spontaneous spasm, the patient experiences other more special and occasional phenomena of a like nature. For example, every now and then there is a sudden spasm of the throat, including the larynx and pharynx, which for a time impedes respiration and causes temporary aphonia. Again, occasionally there is a sudden contraction of both orbiculari palpebrarum muscles, so that both eyelids are tightly closed for half a minute or so. The cutaneous reflexes can everywhere be elicited, but are not exaggerated.

The most noticeable feature of this case is the remarkable increase of muscular contraction which follows percussion of the tendons, the muscles themselves, and the bones. From above downwards the most prominent phenomena were as follows. A tap on the bone over any portion of the frontal or temporal regions caused at once tonic contraction of the orbiculari palpebrarum muscles, immediately followed by a clonus, in which the eyelids were opened and shut with great rapidity. This lasted for perhaps half a minute, when it somewhat suddenly stopped, to be again produced by another tap on the bone in the neighbourhood. This phenomenon was preceded by fixation of the eyeballs in an upward direction. On the mouth being opened, one finger of the observer was placed on the teeth of the lower jaw, and when this was tapped, a well-marked jaw-jerk was obtained. If the pressure was kept up, a rhythmical clonus ensued, which continued as long as the tension was maintained. This clonus was large in degree and slow in rhythm, being about 6 or 8 to the second. At other times if the lower jaw itself was tapped with the percussion hammer, a very fine, rapid, rhythmical tremor of the jaw was produced, roughly speaking about 15 or 20 to the second, which continued for a few seconds and ceased by itself. Occasionally the artificially produced jaw-clonus was followed by temporary spasm of the larynx and pharynx, in which there was inability to breathe for some seconds, and suffusion of the face, a feeling of oppression and lump in the throat, and inability to speak. This aphonia lasts for some minutes afterwards. Percussion of all the tendons throughout the body causes the most violent manifestations, in the shape of an immediate jerk of the muscle operated on, which is at once succeeded by clonic trepidation of the entire limb. Frequently the local convulsion spreads thoughout the entire body. For example, a slight tap on the patellar tendon produces a violent extension of the leg, at once succeeded by clonic shaking, of a large type, of the whole lower extremity, after a second or two of which the back stiffens, the head is bent backwards, and a condition of opisthotonus ensues, with rigidity and trepidation of the head, neck, and all the extremities. This lasts for 10 or 12 seconds, and then suddenly relaxes. The same ensues when any tendon is percussed, especially those of the left arm and right leg, and also when a blow is given to any of the prominent bones, or a jar to any part of the body is received. This spasm is not accompanied by actual pain, but the sensation is unpleasant and arrests the respiration. There is no loss of consciousness during these paroxysms, the patient being perfectly acute intellectually, and able to describe her sensations. Ankle-clonus cannot be elicited; but when vigorous attempts are made to do so, there is a tendency for universal spasm to be produced. Response of muscles to mechanical percustion lively, but not excessively exaggerated. Sensibility is everywhere natural. There are no trophic changes, and the electrical reactions of the nerves and muscles are normal.

The patient was kept for a few days without treatment while the foregoing observations were being made. She was then directed to lie in bed, and ordered chloral hydrate, gr. xx.; pot. bromid. gr. xxx., every six hours. Fourteen days afterwards all the symptoms were notably diminished in degree, but still preserved the same characters as before. About this time she was seized with a mild attack of pleurisy of the left side. The draught prescribed above was discontinued, and poultices were applied to the chest. In one week she was convalescent from the pleurisy, and in two had quite recovered. It was then found that all her nervous symptoms had disappeared. On getting out of bed she felt weak, but after a few days expressed herself quite well. There was no trace of spontaneous spasm. The gait was normal, and the patient moved about the ward all day without fatigue. No clonus could be elicited either of eyelids, jaw, throat, upper or lower extremities. Everywhere percussion of the tendons produced natural responses, except at the knees, where they were somewhat lively, but not excessively so. The patient, when seen a month after discharge from the hospital, was found to have retained her improved condition.

Commentary.—This woman, as far as could be ascertained, was healthy in every respect except in the particulars just described, namely an abnormal readiness of all the muscles of her body to contract on the application of local percussion to their substance, and especially to their tendons; and also for this contraction to be succeeded by a rhythmical clonus, sometimes located in the individual muscle, and sometimes spreading to the entire limb or throughout the whole body.

The nature of the morbid process which led to the production of these symptoms is very obscure. That it was not the result of serious gross organic disease is proved by the fact that, although of two years' standing, the affection completely recovered in a few weeks. In the absence of a positive anatomical demonstration we are reduced to speculation, and may theoretically assume the position to be most plausibly explained by a condition of extreme functional over-activity of the ganglion cells of the anterior cornua of the cord and medulla, causing hyper-tonicity of the entire muscular system and its consequent tendency to tonic and clonic spasm. Even granting this to be the proximate cause of the physical phenomena, we have in this case no means of knowing whether it was primary or secondary to changes elsewhere. There was certainly no evidence of the latter.

The spontaneous tonic spasms, the increased tendon reflexes, and the rhythmical clonic trepidations have all probably the same physiological signification, and their occurrence in different parts of the body is not of special pathological import, but depends only on the distribution and extent of the morbid process to which they owe their origin. The jaw clonus, for example, is not characteristic of any particular disease, but only implies involvement of the medulla or those central regions the irritability of which leads to hyper-tonicity of the muscles which move the inferior maxilla. The symptom is not so rare as seems to be supposed, and doubtless, when looked for, it will be found more commonly. I have repeatedly demonstrated it in cases of disseminated sclerosis, spastic paralysis, and in so-called hysteria. Others have also observed and recorded this symptom.[1] Although in itself of no special significance, jaw clonus, being one of the highest available deep reflexes that can be obtained, is on that account of great clinical importance. The same applies to what Dr. de Watteville calls "the jaw-jerk," a condition which has already been described by Dr. Lewis of Philadelphia, under the term "Chin-reflex.[2]

In this case the jaw-jerk was well marked, as also was the clonus, when pressure was kept up by the finger on the teeth of the extended inferior maxilla. In addition, however, to this ordinary clonus of six or eight contractions to the second, a tap on the masseter or on the lower jaw, not necessarily in an extended condition, caused another form of tremor, consisting of a fine, rapid, rhythmical clonus about twice as fast as that produced by continuous extension, and this lasted for ten or twelve seconds after the percussion blow, and then ceased of its own accord. Similar phenomena followed a tap of many of the other muscles of the body, and also from a jar of the bones in their neighbourhood. This was notably observed in the orbiculari palpebrarum and oris, in the other muscles of the face, in the muscles of the neck and larynx, and in some of the small and superficial muscles of the trunk and limbs, although in these latter they were complicated with the larger clonus and tonic spasm. This fine rapid trepidation, the result of a direct blow or general jar, was very similar to what is seen during a rigor; the chattering of the teeth being almost identical in both. The condition necessary for its production is probably an exaggeration of that necessary to produce ordinary clonus; the greater rapidity of the tremor being possibly due to its velocity not being impeded by passive flexion of the muscles. The two phenomena may, on the other hand, depend on entirely different mechanisms. Further observations on the subject are necessary to illuminate the question; and these may throw light on many obscure points in connection with rigors, shiverings, tremblings, and such-like conditions.

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[1] Ballet, "Note sur l'état de la réflectivité spinale dans la fièvre typhoïde;" 'Prog. Méd.,' 1881.

Delom-Sorbé, 'De la trépidation épileptoïde provoquée.' Bordeaux. 1885.

Weir-Mitchell and Lewis, "Physiological Studies of Knee-jerk," &c.; 'Medical Times,' New York, Feb. 1886.

[2] 'The Chin-reflex. A new Clinical Observation.' By Morris J. Lewis, M.D., Medical and Surgical Reporter. Philadelphia: May, 1885.

The attacks of tonic spasm with distortion of the limbs in this case are also of interest, and not easy of explanation. In some respects they resemble a condition described and illustrated by myself in a former number of 'BRAIN.'[1] Those seizures of tonic spasm were usually spontaneous, arising without apparent exciting cause at irregular intervals. On the other hand, it is to be noticed that the attacks of clonic trepidation already described rarely did so, but almost always had to be artificially provoked. It is possible to conceive that the same pathological basis may be the origin of these two conditions. Assuming a continuous irritation of the central nerve-elements to be the cause, the accumulation of motor energy exploding at intervals might give rise to the spontaneous attacks. Under the same conditions a mechanical stimulus would originate similar phenomena, and passive stretching of the muscles give rise to clonic spasms. But why the spontaneous seizures should always be of a tonic nature, and those provoked be clonic, furnishes matter for speculation.

The treatment of this case was based on purely hypothetical considerations, and turned out to be successful. Primary functional irritability of the grey matter of the cord having been assumed, hydrate of choral, and bromide of potassium in large doses were administered. These drugs are known in health to depress the functional activity of these centres. Although the affection had lasted for two years, and the patient had previously tried the effects of rest and other methods without benefit, the result of these remedies was immediate, and in a fortnight all the symptoms were notably diminished. An accidental attack of pleurisy seemed to complete "the cure."

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[1] 'BRAIN,' January, 1885.

CASE OF CORTICAL PARALYSIS.

BY J. A. VOORTHUIS, M.D. (AMSTERDAM).

AN orphan boy, 14 years old, of delicate constitution, complained on the 1st of March, 1884, of a sensation of numbness in his left hand and forearm. This fact at first attracted no attention, because other symptoms were totally wanting. In the evening of the same day, however, the patient had three attacks of clonic-spasms in the left arm, accompanied with tossing and shaking of the head. No mental alteration was present. Next morning Dr. d'Ailly, Physician to the Amsterdam Orphanage, whom I have to thank for the clinical observation, noticed paresis of all the muscles of the left arm and leg; the patient still had a sensation of formication. On the third day both members were totally paralysed, and anæsthetic formication in the left foot was now felt. The facial muscles did not present any deviation. Speech, though the patient's articulation had always been a little difficult, was unaffected, and intelligence unaltered. The boy lay on his bed, and played at draughts as well as before. No change took place until the 11th of March, when the patient suddenly dropped down with complete loss of consciousness; profound sopor followed, from which he could be roused only by being loudly spoken to; he then looked up, but could not answer questions.

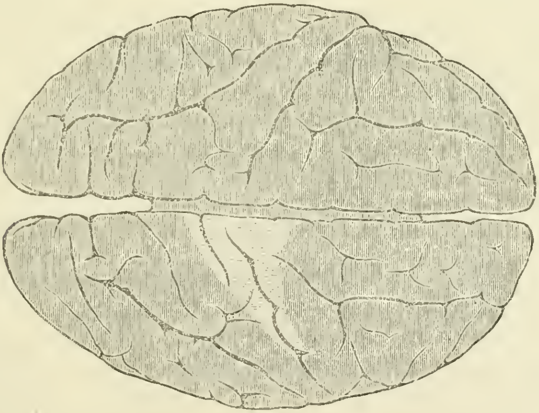
Frequent erections occurred, with incontinence of urine, the bladder being unfilled. Rapidly increasing decubitus appeared in the middle of the sacrum. At first the pupils were unaltered. After the apoplectic fit very sluggish contractions followed the stimulus of light. The reflexes and electrical excitability were not investigated at the time. No contracture, nor obvious vasomotor change, but paresis of all the facial branches on the left side was observed. The tongue when protruded deviated to the right side. The closure of the eyelids was imperfect in both eyes, especially in the left. Sensation in the face, palate and uvula was not tested.

It was noticed that the boy saw better with the right eye.

No headache. Somnolence slowly increased, and the patient died on the 19th of March, in the morning.

Recapitulating the clinical symptoms, two stages can be distinguished:—1. A stage of irritative symptoms, slowly passing into symptoms of paralysis; consciousness and intelligence being unaffected. 2. A stage of deep somnolence beginning with an apoplectic attack.

Autopsy of the head only was permitted. The brain was examined 36 hours after death in the laboratory, and with the aid of Professor Kuhn, whose assistant I was at the time. Skull-cap easily removed, somewhat thin, otherwise normal. Outer surface of the dura mater of a dark reddish colour. Right hemisphere appears somewhat swollen, and less resistant to pressure. In the longitudinal sinus a discoloured clot was found, reaching from the frontal to the occipital extremity. In the midst of the sinus the clot contained a thrombus, 1·5 cm. long and 3 mm. thick, adherent to the wall of the vessel on the right side, but not extending into the lateral branches. No fluid in subdural space. The dura mater was adherent to the pia mater over the central convolutions in the middle of the right hemisphere, over a triangular area presenting a basis of 3 cm. along the longitudinal sinus, and an apex 6 cm. from the base, lying near the fissure of Sylvius. With some trouble the dura could be detached, except over a small patch at the seat of the above-mentioned thrombus. The inner surface of the dura-mater was rather rough and dry. The larger vessels of both hemispheres were strongly filled with blackish liquid blood, and the pia somewhat dry but transparent, studded with some grey tubercles. The part of the



pia involved in the above-mentioned triangle and covering the two central convolutions, was opaque and thickened (reaching 1·5 mm. near sagittal fissure). A greenish-yellow coagulated exudation around the vessels, and a few minute hæmorrhagic points were seen. Basis cranii normal. On the basal aspect of the cerebrum nothing was noticed but adherences in the fossæ Sylvii, caused by the growth of numerous granulations, especially right side. The arteriæ fossæ Sylvii were unaltered. After stripping the pia mater, the convolutions on the right side were somewhat flattened and flaccid. The corpus callosum on the right side was somewhat weakened; on opening the ventricles a small quantity of colourless but turbid liquid escaped. The ependyma in both ventricles was smooth and bright. The centrum ovale on the right side was softened and quite white, presenting no puncta cruenta on section. Further examination showed that on the above-mentioned triangular area the pia mater and cortex cerebri were coherent; the same was noticed over the gyrus fornicatus and gyrus paracentralis; sections of these convolutions displayed some hæmorrhagic points both in the pia and cortex cerebri. In the fissure of Rolando a granular mass as large as a pea was found, with a reddish colour and dark hæmorrhagic points; the cerebral tissue about it had a reddish colour and more dense. In the cortex of the central convolutions as well as in the neighbouring portion of the corona radiata, numerous hæmorrhagic points were visible. Dissection of the basal ganglia, pons, medulla oblongata, cerebellum, did not show any anomaly.

Microscopical examination confirmed the diagnosis of tubercular growths in the pia-mater, and showed in several perpendicular sections of the central convolutions a new formation of very small vessels, distended with blood; and an infiltration with rather large cells with almost invisible nucleus.

To sum up: there was a general tuberculous eruption in the pia-mater, with local exudative meningitis involving the cortex cerebri of the upper part of the central convolutions, the gyrus paracentralis and fornicatus; subsequent thrombosis of longitudinal sinus, causing important venous hyperæmia; hæmorrhage in the inflamed region, and softening of the whole right hemisphere.

In both the clinical and the anatomical factors two distinct stages can be distinguished, which have obviously mutual relationships; and so I believe to be right in attributing the symptoms of irritation in the left extremities, and the subsequent paralysis, to the inflammation proceeding from the meninges, and involving the cortex cerebri in the Rolandic region. The fact of the disease beginning with paræsthesia and the anæsthesia appearing on the third day, are points of especial interest.

The second stage in the clinical history, namely the apoplectic fit with subsequent coma, is accounted for by the hæmorrhage, and softening of cerebrum. Although this case is somewhat complicated, it may fairly claim some of the physiological value of an experiment performed by nature, illustrating in a distinct manner the motor functions of cortical centres.

PARALYSIS OF THE ARM FROM LESIONS OF THE NERVE TRUNKS.

BY ALEXANDER M. M'ALDOWIE, M.D.

THE following cases present no uncommon nor obscure pathological features, but they appear to me to form an interesting group from a clinical point of view, and worthy of record. Not only are they all cases of external paralysis, but in all the loss of power was the result of injuries to, or pressure on, the nerves, so that no difficulty was experienced in detecting the cause, nor in selecting the appropriate remedy.

CASE I.—William H., æt. 55, a collier, came under my care on December 23, 1884. Nine weeks before, the patient's arm and side were injured in the pit by the falling of a mass of coal. The arm was severely crushed, and there were three lacerated wounds on its anterior and outer aspects, about four inches above the elbow-joint. Immediately after the accident, there was complete loss of motion and sensation in the limb below the seat of injury. Sensation began to return in about a week, but the paralysis remained.

At the date of examination the forearm was completely paralysed, the muscles flaccid, the hand slightly flexed with wrist drop. There was no atrophy of the muscles. Sensation was normal. The muscles responded slightly to electrical stimulation. The wounds were healed, but there was great tenderness on pressure over the cicatrices. Patient complained of shooting pains down the arm and forearm. Iodide of potassium was adminished internally, with galvanism and friction to the affected limb. On the 5th of January, he was able to move his fingers; and on the 9th, had slight power of movement in the muscles of the arm and forearm. From that time progress was rapid and he left off coming about a month later, having quite recovered the power in the forearm, although the muscles of the arm were weak and stiff from the large cicatrices.

In this case all the nerves in the arm were affected. The nerve-trunks must have been crushed by the accident, not severed by the wounds, as sensation returned within a week. Moreover, there was no wound over the tract of the musculo-spiral, and yet the muscles supplied by it were as completely paralysed as the others.

Case II.—Mrs. M., æt. 33, a widow, came to consult me on the 22nd of August. Seventeen days before, she went to sleep sitting on a chair. She woke up during the night suffering acute pains in the left arm, which was hanging over the back of the chair. The pain was situated about the middle of the arm on its inner aspect, and the limb was quite cold and numb. She was unable to move the arm in the least for more than an hour, when she recovered the power of flexion. Sensation also speedily returned.

On examination, the extremity presented well-marked symptoms of paralysis of the musculo-spiral nerve. Tested with the dynamometer the power of grasping with the left hand was 15 lbs., whilst with the right the power was 45 lbs. The hand felt quite warm. There was no loss of sensation, and no atrophy of the affected muscles, which reacted normally both to the faradic and galvanic currents.

The treatment consisted of strychnia internally, stimulating liniment and friction to the arm, and galvanism daily. Progress was gradual but satisfactory, as she had complete recovery in less than two months.

This was an ordinary case of paralysis from compression, and does not call for further remark here.

Case III.—Mrs. F., æt. 32, came to me complaining of pains and cramps in the right arm and hand. The pains came on suddenly a fortnight before, during labour. She took no notice of them for some time, but, as they continued to increase, she came to me for advice.

The pains were severe and lancinating, shooting down the front of the arm and forearm to the fingers. The limb presented exactly the appearance described by Trousseau as characteristic of tetany. The forearm was flexed, the thumb adducted, the fingers pressed together and semi-flexed, so that the hand assumed the conical shape which is pathognomonic of that complaint. The muscles were stiff and rigid. There was hyperæsthesia of the skin and the affected region. On attempting to extend the fingers, violent and painful spasms were produced. A firm circumscribed swelling could be felt in the axilla in the tract of the axillary artery. It was very tender on pressure, and any attempt at manipulation induced the painful spasms in the forearm and hand. Pulsation in the artery below the swelling, and at the wrist, was almost imperceptible. The heart was greatly hypertrophied, and a loud systolic murmur was heard at the apex. The patient stated that she had an attack of rheumatic fever some years before. Belladonna and opium liniment was applied to the swelling in the axilla and to the affected muscles, and iodide of potassium administered internally.

About ten days afterwards she came again for examination. The pains had almost quite left. The spasms were gone, leaving the flexor muscles flaccid and paralysed. The fingers were extended, the palm flat. There was no loss of sensation. The muscles responded to electrical stimulation. There were still tenderness and thickening in the axilla. The diagnosis I made at the time was: embolism of the axillary artery at the point where the two heads of the median cross, producing at first irritation and afterwards paralysis of the nerve. The patient never returned for advice, and, as I had neglected to note her address, I unfortunately was unable to learn the further history of this case.

Case IV.—James H., æt. 8½. Patient was knocked down at school three years before, spraining his left shoulder. A few weeks afterwards, a swelling formed in the arm-pit. This remained hard and painful for three or four months, when it suppurated, and there was a slight discharge, but the tumor remained up to the date of examination. Four months previously it again became inflamed, but did not suppurate. A month ago the boy began to suffer from pain and spasm in the flexor muscles of the forearm and hand. The spasm has continued with more or less severity ever since. Unable to use the hand; occasionally cries from the intensity of the pains and cramps in the arm.

On examination. — Child pale and ill-nourished. No diarrhœa. Fingers slightly flexed and pressed together. Thumb firmly pressed against the palm, producing an indentation on its surface. The skin on the parts of the thumb and palm which were in apposition was white and soft, and bathed in sour perspiration, showing that the pressure must have been long and continuous. Flexors of forearm contracted and rigid. A glandular enlargement about the size of a walnut occupies the outer boundary of the axilla. Painful spasms were induced by attempting to straighten the fingers, and by pressure over the front of the elbow or over the tumor in the axilla. Sensation appeared to be normal, but the child was frightened and refused to answer questions. Quinine was given internally: the hand and arm to be bathed twice a day, with friction to the affected parts, and the continuous current daily. Iodine ointment rubbed gently over the swelling. Patient improved steadily under the treatment, and was quite well in three weeks.

This was a typical case of tetany, caused by pressure on the median nerve.

Case V.—Edward W., æt. 22, forgeworker. A month before examination the patient fell from a height on his right hand. The wrist was flexed, so that the whole weight of the body came on the dorsal surface of the hand. He was unable to extend the fingers after the accident, and has had paralysis of the extensors ever since. There was no loss of sensation.

On examination.—Hand kept half shut, wrist-drop, unable to extend or separate fingers. Sensation normal. Extensors react slightly to electrical stimulation. No atrophy of muscles. No tenderness nor pain in any part of the arm or forearm. No adhesion, nor symptoms of injury to the tendons or wrist joint. Patient quite able to move the elbow-joint. The treatment consists of the hypodermic injection of strychnia three times a day, iodide of potassium internally, galvanism and friction to the affected part. He remained under treatment for over a fortnight, and then left for his home in Yorkshire, having recovered slightly the power of extension.

The etiology of this case is somewhat more obscure than in the preceding ones. In my notes of the case I have entered the diagnosis as injury to the musculo-spiral nerve. But the late Professor Pirrie often pointed out to his students, that in primary amputations for injury the muscles frequently failed to contract, and he held that this was due to molecular injury of the muscular fibres from overstrain. It is therefore possible that a similar condition might have occasioned the symptoms noted above.

The cases which I have narrated show either exaltation or diminution of function produced by pressure on the nerve trunk. Weir Mitchell has shown, that a pressure of from 18 to 20 inches of mercury is sufficient to check motor impulses completely, and also to destroy the medullary substance of the nerve.

"Slight mechanical pressure often leaves no perceptible traces in the nerves, no change of the electric excitability, and no secondary trophic disturbances," says Erb; "yet very obstinate paralysis often follows such pressure, as is seen, for example, after pressure applied to the musculo-spiral nerve, and it is impossible to deny that even a slight mechanical action may so change the molecular condition of the motor nerves as to abolish their power of conduction."

These cases afford striking evidence of the well-known fact, that if mixed nerves be exposed to pressure the motor filaments suffer much more injury than the sensory. The following case illustrates this even more forcibly.

Case VI.—John C., aged 36, potter, Jan. 1, 1881. Ten days ago was working with his arm dipped in cold water when it suddenly became quite powerless, and he found that sensation was also totally lost. Sensation returned in a day or two, but there has been only slight recovery of motion. The hand and forearm are now almost completely paralysed. Tested with the dynamometer the power is ten pounds. No atrophy of muscles. No loss of sensation. The hand feels cold; the patient rapidly recovered power of motion and was able to resume work on the 16th of January. In this case cold, the exciting agent, must have acted primarily, and to a much greater extent on the sensory filaments than on the motor, and yet the former recovered their function much more rapidly than the latter.

None of the symptoms enumerated in these cases indicates neuritis, although a slight amount of inflammation might have been present in some. Even electrical examination yields no infallible test of the presence or absence of inflammation, for in slight neuritis no change whatever may be found in the electrical reactions. Whilst authorities agree that no single symptom is pathognomonic of inflammation, yet the following undoubted case of neuritis from pressure, now under my care, presents a marked contrast to the above.

Case VII.—George S., æt. 57, has been employed as a gilder in the potteries for more than forty years. In his occupation he uses a small board to steady his hand, resting his arm on it, so that the edge presses against the inner side of the arm just above the elbow, and also against the upper part of the inner aspect of the forearm. Three months ago he began to suffer from pains and cramps along the inner side of the forearm, with numbness and tingling of the skin. He described the feelings as being exactly similar to those produced when the ulnar nerve is pressed behind the inner condyle. I found tenderness over the ulnar nerve both above and at the elbow; pressure causing shooting pains up the arm, and also down to the little and ring fingers. There was well-marked atrophy of the muscles of the ulnar side of the forearm, diminished galvanic and faradic reaction, and considerable loss of sensibility. This case is probably one of chronic interstitial neuritis resulting from slight and long continued pressure. It differs from the others chiefly in having well-marked trophic changes and diminished sensibility. The prognosis is hopeful, as some years ago he had a similar attack, which passed off after rest and appropriate treatment.

For the notes of the following case I am indebted to Dr. Hind, of Stoke-on-Trent, with whom I saw the case in consultation.

Case VIII.—M. S., æt. 7 months, said to have been injured in birth. The nurse states that the confinement was very hard and that the doctor had to turn. Both arms got lodged above the head, the left was broken, and the right hung useless. For five months the right arm did not grow at all, but the left soon got well: otherwise the child came on well. When first seen (two months ago) the right arm hung useless at the side. No sensation, no motion. There was no muscular rigidity. The first phalanges were extended on the metacarpals and the second and third phalanges flexed. The palm of the hand appeared elongated from the extension of the first phalanges. The thenar and hypothenar eminences were wasted. The ulna and radius above the wrist were very small and soft, and the joint very loose. The child was well developed both mentally and bodily. A faradic current of moderate strength was applied, but it was neither felt nor did it cause muscular contraction. There was no evidence of tumour nor callus pressing on the nerves. Electricity was applied every two or three days; one pole (−) at the cervical spine, the other (+) moved over the various muscles. In about seven to ten days very slight movements were noticed: and the child seemed to have a little power in the pectorals, and now and then there was a slight movement in one of the flexors.

Present condition. These movements are now more general, and the child attempts to raise the arm. The child was observed on one occasion to extend the index finger completely and then close it. The size of the forearm has increased, and the bones have become larger and more solid. It is not certain whether the current causes any "pins and needles," for sometimes with a powerful current the child will not exhibit any expression of sensation, whilst at others she cries. But a needle-prick produced no cry of pain or appearance of feeling. The skin looks quite healthy.

Treatment. Electricity and friction.

These cases in my opinion form an interesting group, showing the different features peripheral paralysis may exhibit, although the exciting cause be somewhat similar in all.

Buzzard remarks:—"In cases of paralysis, in which the lesion certainly occupies the peripheral nerves, we may find a singular diversity of symptoms. Pain is sometimes present and sometimes absent, numbness may be slightly or strongly pronounced, muscular atrophy, which is sometimes conspicuous, may be entirely wanting, whilst the results of electrical examination may vary to a remarkable extent."

It is interesting to notice that in the two cases in which tetany was observed, one occurred in childhood, the other during the puerperal state, both of which are mentioned by Trousseau as predisposing causes.

The prognosis in every case was favourable. In all recovery was either complete, or the power of motion partially restored while under my care.

Critical Digests.

AMYOTROPHIC LATERAL SCLEROSIS.

BY J. A. ORMEROD, M.D., F.R.C.P.

There are many ways in which this disease is interesting and remarkable—in its clinical characters, which combine spinal with bulbar symptoms, and the phenomena, usually opposed to each other, of atrophic paralysis with spastic condition of the limbs; in its relation to the large and vague group of muscular atrophies, from which it stands out as a definitely distinguishable type; in its pathology, which offers an excellent example of a "systematic" or "tract" disease.

It is to Professor Charcot that we are indebted for the first description of the disease in its most typical form.[1] This description is now so well known, that we need only recapitulate here the main points of it. Amyotrophic lateral sclerosis resembles in many points, and indeed was originally confounded with, the progressive muscular atrophy described by Cruvelhier, Aran and Duchenne, and Roberts. That which is common to the two diseases is a paralysis with atrophy of muscle, often accompanied with fibrillary twitchings, which begins usually in the hands, or at least the upper limbs, is progressive in character, is not limited to the district of particular nerves, and is not accompanied by anæsthesia or marked sensory phenomena. But amyotrophic lateral sclerosis presents the following peculiarities. Its course is more rapid, for death usually results within three or four years; it involves the muscles of the limbs both more rapidly and more uniformly; thus although wasting may commence in the thumb-ball, it spreads before long to the whole mass of muscles of the hand, forearm, or even upper arm; the wasting also is preceded by a stage of weakness out of proportion to the then loss of muscle; paraplegia, though perhaps without wasting, sets in early in the legs. Further, and this is most important, the condition of the limbs is not, as in most forms of atrophic paralysis, lax and flaccid, but is accompanied by contractures, appreciable indeed even in the wasting arms, but still more marked in the legs, where paralysis predominates over atrophy. Lastly, at some stage of the disease, usually a late stage, bulbar phenomena appear, with a regularity unknown in the disease of Aran-Duchenne. A typical case, in fact, presents three stages: (1) weakness, soon followed by wasting and actual paralysis of the hands and arms; (2) paralysis, usually of the spastic type, of both legs; (3) the appearance of bulbar phenomena.

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[1] 'Leçons,' vol. ii. p. 227 foll.

As to the morbid anatomy, we shall only say for the present that in addition to the atrophy of the anterior cornual cells which is the essential lesion (in some cases at least) of progressive muscular atrophy, there exists also a fasciculated sclerosis of the lateral columns of the cord; and that in his earlier writings, Charcot correlates the two things as follows: the lateral sclerosis is the initial lesion, and causes alike the primary paralysis and the contractures; while from the lateral columns (following up their physiological and anatomical connections), the disease passes to the cells of the anterior cornua, and causes, by their destruction, the muscular atrophy.

As an illustration conforming in many respects to the type described by Charcot, we will give in brief abstract a case recorded by R. J. Collyns, in the 'Reports of St. Bartholomew's Hospital' for 1883.

This is the case of a man, aged 32. The total duration of the disease was very short, only eleven months in all; he was admitted seven months after its onset. He had been habituated to smoking and drinking. The symptoms before admission had been as follows: Loss of power in the left, then in the right hand, then in the forearms. Nasal twang in the voice. Wasting of the upper limbs; shaking of the legs, stiffness of the knees, and dragging of the legs when he walked. Muscular twitchings. Inability to frown or to hold a cigar in his mouth. Difficulty of deglutition, hoarseness, salivation, attacks of dyspnœa. He had been also subject to fits of uncontrollable laughter.

His state on admission was this. His voice was nasal and monotonous. There was wasting of the facial muscles, and an expression of laughter when he tried to speak. The tongue was wasted, but showed no vermicular movements. The palate was partly paralysed, and the muscles of mastication were weak. There was serious difficulty of swallowing, but this improved. There was muscular wasting about the trunk. In the upper limbs there was wasting, most marked in the hands and forearms; no rigidity, except so far that "the hands were always pronated, and it required some force to supinate them." The lower limbs were less wasted; the patellar-tendon reflexes were exaggerated, and there was ankle-clonus on both sides. His gait was spastic. The electro-irritability of the muscles, both faradic and galvanic, was diminished in proportion to the atrophy; the polar formulæ are not mentioned.

During the first two months of treatment (strychnia and electrisation were employed) there was considerable improvement. After that, the atrophy and paralysis began to progress rapidly; rigidity of the calves developed; much difficulty in articulation appeared; attacks of dyspnœa and dysphagia; the facial expression changed and became lacrymose. Death resulted from hypostatic pneumonia and collapse of the right lung.

The changes found in the nervo-muscular system were as follows:—

Muscles.—Some fibres had lost their transverse striation; others were granular, hyaline, or longitudinally striated. Many fibres were small. There was increase of the interstitial connective tissue.

Nerve-trunks.—No well-marked pathological change. In the *cord* there was sclerosis, most marked in the crossed pyramidal tracts. but noticeable also in the direct pyramidal tracts, and in the anterior columns. In the cervical region the sclerosis of the lateral columns extended forwards as far as the outer angle of the anterior cornua; in the dorsal region it reached forwards only so far as an imaginary line drawn transversely through the posterior commissure. In both cervical and dorsal regions, though not in the lumbar, it reached right up to the posterior cornua. It was most intense in the area of the crossed pyramidal tracts, and in the lumbar region was limited to this area. The cerebellar tracts were intact throughout. The sclerosis was characterised by increase of the neuroglia and atrophy of the majority of nerve fibres.

As to the grey matter, the anterior cornua were diminished in size, and their nerve cells were atrophied: this change was most marked in the cervical and dorsal regions, though present also in the lumbar. The cells of Clarke's columns were comparatively, though not wholly, intact.

In the *medulla* the sclerosis could be traced up through the pyramids into the pons, but not beyond this. It is noted that immediately below the decussation of the pyramids the direct pyramidal tracts were normal, though diseased lower in the cord. The motor nuclei in the floor of the 4th ventricle were defective, and their cells atrophied.

The author justly remarks upon this case, that though all the symptoms described by Charcot were present, yet that the symptoms referable to disease of the grey matter, viz. muscular atrophy and loss of electro-irritability, predominated, and he thinks it probable that the disease may have begun in the grey matter rather than in the lateral columns. In this respect, then, the case is not wholly typical, but it will serve as a transition from Charcot's type to that which we shall next notice.

This is a class of cases fully described by Leyden[1] under the name of "progressive bulbar paralysis;" apparently he considers them to differ essentially from those described by Charcot. The symptoms are here those of a glosso-labio-laryngeal palsy, associated with muscular atrophy of the limbs. The most prominent and usually the earliest phenomena are those of bulbar lesion.

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[1] In 'Archiv für Psychiatrie,' &c., vols. ii., iii., viii.; and in 'Klinik der Rückenmarkskrankheiten.'

Paralysis, generally insidious, of the tongue, lips and soft palate, sets in, manifesting itself first in disturbance of the functions of articulation and deglutition; next, in actual loss of power in the tongue accompanied by wasting and fibrillary tremors. The saliva runs from the mouth; the lower part of the face becomes lax, immobile and lacrymose; contrasting with the play of the forehead and eyes, which remains natural. After these symptoms (referable to disease of the hypoglossal and facial nuclei) come others in the district of the vagus and accessorius nerves, viz. hoarseness and aphonia, attacks of dyspnœa, and of an anginal nature. The muscles of the neck may become weak and atrophied, and sooner or later supervenes progressive muscular atrophy of the hands, arms, trunk and lower limbs. Death usually comes in an attack of dyspnœa, or food may enter the air-passages and cause pneumonia. The disease rarely lasts more than three years, though Leyden has seen the symptoms remained unaltered for seven.

Post-mortem, there is found atrophy of the muscles, degeneration of fibres in the nerve-trunks, and of the anterior nerve roots, and of some of the cerebral motor nerves; a corresponding atrophy of the spinal motor cells (especially in the cervical region), and of the motor nuclei at the lower part of the 4th ventricle, more especially of the hypoglossal nucleus. But in addition there is degeneration, in the cord, of the pyramidal tracts, crossed and direct, which can be traced sometimes up through the medulla into the pons; there is degeneration also, though less intense, in certain other parts of the lateral and anterior columns, especially in the cervical region.

Now we shall see that these are exactly the lesions which are found in amyotrophic lateral sclerosis.

Let us consider, then, the clinical difference between the two diseases, and see whether they are sufficient to outweigh the pathological resemblances.

One striking clinical difference is the predominance (in Leyden's disease) of bulbar symptoms, and their presence from the commencement. Against this we must note, that muscular atrophy of the limbs *may* be the earliest symptom of a progressive bulbar paralysis; and that, on the other hand, Charcot has expressly described a variety of amyotrophic lateral sclerosis which begins with bulbar symptoms.

The second difference is that, according to Leyden's description, the paralysis and atrophy proceed pari passu, whereas Charcot says that in amyotrophic lateral sclerosis there is a stage of motor weakness which precedes the atrophy.

The third and most important difference is with respect to spastic phenomena. These constitute for Charcot an essential symptom; since they are to be regarded as the expression of the lateral sclerosis, the existence of which differentiates the disease from ordinary progressive muscular atrophy. But Leyden asserts most positively that his paralysis is of the flaccid type. He says,[1] "Spastic symptoms, such as have attracted so much attention lately, are absent in our type of the disease, or present in very slight degree. In particular, the symptom of rigidity of the muscles has not been observed, nor clonic tremor of the leg-muscles; the so-called spinal epilepsy, and abnormal tendon-reactions do not occur, and contractures are either absent altogether or present only in a limited sense. No rigidity or contracture is observed in the face or the neck-muscles. In the hands, arms, and feet, however, contractures do occur, and stiffness of the vertebræ is once mentioned. These contractures, however, do not originate in spastic (irritative) phenomena, but their essential source seems to be in the habitual position of the limbs. to which the muscles adapt themselves and resist alteration of their position. Contracture of the hands and fingers leading to the claw-hand is most frequent. . . . Similar stiffness may occur in elbows or shoulders; club-foot may arise, or stiffness of the knee-joint, when the patient sits long. Several of these contractures seldom occur together; they are mostly slight and often absent together."

Charcot indeed contends,[2] and we think with reason, that in these and other minor phenomena recorded by Leyden himself are to be found the evidence of a spastic condition; and secondly, that the tendon-reactions, those most delicate tests of such a state, were not properly investigated by him. We do not, as a matter of fact, find in Leyden's descriptions a systematic account of the tendon phenomena; nay, in a case of bulbar paralysis quite recently recorded,[3] it seems to have happened unaccountably that the tendon-phenomena were not noted. But after taking all this into account, the broad fact remains, that in the one clinical type spastic phenomena form a striking and almost constant feature; in the other they are, if not absent, unobtrusive and liable to be overlooked.

The explanation of this fact may, we think, be found if we are allowed to take a rather broader view of the pathology of this disease than that propounded at first by Charcot. If, as he supposes, the disease always begins in the lateral columns and spreads thence by a sort of physiological continuity to the motor cells, and if, as is generally supposed, the disease of the lateral columns finds its clinical equivalent in spastic phenomena, then these must evidently constitute early and essential symptoms. But let us assume that the disease, though limited or mainly so to the motor tracts, may begin at any point in those tracts, and spread in any direction through them. Then there will be cases in which the grey matter is first attacked, and in these there will be atrophic paralysis, but not necessarily contractures. Indeed, since Charcot has shown that in the late stages of a typical case when the grey matter has been deeply involved contractures disappear, we should imagine that if this part is attacked first they would be absent altogether.

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[1] 'Arch. d. Psych.' vol. viii. p. 650.

[2] 'Leçons sur les localisations cérébro-spinales,' p. 401.

[3] Freund, 'Deutsch. Arch. f. klin. Med.' 37, p. 405. Abstracted in 'Neurolog. Centralblatt,' vol. iv. p. 510.

The same supposition would explain the absence, in Leyden's type, of the stage of pre-atrophic paralysis.

But why, it may be asked, should this primary affection of the grey matter occur especially in the bulbar cases? This is difficult of explanation; but an examination of cases will show that no hard-and-fast line can be drawn in this respect between cases originally bulbar and originally spinal. Thus in a case by Maier,[1] which began with bulbar paralysis, then developed afterwards clonic muscular spasm, then double equino-varus and contracture of one hand. In a case recorded as labio-glosso-laryngeal palsy by Hun,[2] there were contractures of the flexors of the hands and arms. On the other hand, in cases primarily spinal, spastic phenomena may be more or less in the background. Thus in addition to Collyn's case quoted above, we note a case by Moeli,[3] in which there were no bulbar phenomena, and there was ankle-clonus but no contractures; again, a case by Vierordt,[4] without bulbar symptoms, in which the author thinks both clinical and anatomical evidence pointed towards a primary affection of the grey matter. There are therefore intermediate cases which tend to bridge over the gap between Charcot's and Leyden's type, and to show that clinically they are, what their morbid anatomy indicates, varieties of one and the same disease.

There are some topics requiring further notice before we pass to the pathology of the disease.

The *etiology* is practically unknown. Exposure to cold, mental or bodily strain, injury, have been cited as exciting cause. Either sex may be attacked. Neither syphilis nor heredity can be set down as predisposing causes. Notable exceptions are, however, recorded by Seeligmüller.[5] He has published five cases of what he believes to be amyotrophic lateral sclerosis in children. Four of them were in the same family. These cases were, however, in other respects exceptional, and no opportunity occurred for post-mortem verification. The average *duration* is about three years, and this period is so seldom exceeded that a bad prognosis must be given in any case. Nevertheless (in addition to one of Seeligmüller's cases which had lasted twelve years), Leyden witnessed a case which remained unchanged for seven years, and one of Dumenil's lasted fourteen years.[6] The *onset* in some of the bulbar cases may be so sudden as to resemble hæmorrhage. In the limbs the distribution of the palsy may be at first hemiplegic, so as to cause confusion with cerebral paralysis unless the state of the muscles be carefully examined. In a remarkable case of Leyden's,[1] the prodomata were those of tabes dorsalis, and indeed a certain amount of posterior sclerosis was found post-mortem. *Pains* may occur at the onset or in the course of the disease, and perhaps some paræsthesiæ in the upper limbs, and back of neck. The *facial expression* varies somewhat according to the type which the disease affects. In both types there is paralysis of expression in the lower part of the face; but in progressive bulbar paralysis the muscles are lax and the expression sorrowful, whereas in cases by Charcot, Gombault, and Pick[2] the face has been drawn into a fixed grin by the muscular spasm. In connection with the tension of muscles of this region may be mentioned the "jaw-clonus" described by Dr. Beevor.[3] The *electrical reactions* exemplify, according to Erb,[4] the "middle form of reaction of degeneration." That is to say, while the faradic irritability of muscle and nerve trunk, and galvanic irritability of nerve trunk remain unaltered, the galvanic irritability of muscle is modified; quantitatively, it is increased or at least not notably lowered; qualitatively the polar formula is altered ($KSZ < AZS$). The same thing holds for progressive muscular atrophy and bulbar paralysis, except that here the galvanic irritability of muscle is diminished; but demonstration of this fact is difficult, on account of the small area of muscle affected. In either disease, when wasting becomes extreme, the electro-irritability disappears. Erb's statements must certainly be received as authoritative; but nevertheless there are discrepant observations by other authors. Thus Berger[5] records that the electrical reactions were normal in every respect; Kahler and Pick,[6] mostly a simple quantitative diminution of excitability, though in some less atrophied muscles $KOZ > AOZ$; Moeli,[7] simple diminution in proportion to the muscular wasting, the polar formula being normal.

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[1] Virchow's 'Archiv,' vol. 61.

[2] 'American Journal of Insanity,' October 1871. Abstract in 'Centralbatt für die Medicin. Wissenschaften,' 1872, p. 429.

[3] 'Archiv für Psychiatrie,' vol. x.

[4] 'Archiv für Psychiatrie,' vol. xiv.

[5] 'Deutsch. Medic. Wochenschrift,' 1876, Nos. 16 and 17. 'Jahrbuch der Kinderheilkunde,' 1878, p. 256. Gerhard's 'Handbuch der Kinderkrankheiten,' vol. v. Abth. i. 2nd half, p. 167.

[6] 'Klinik der Rückenmarkskrankheiten,' ii. 441.

The *diagnosis* is not likely to be difficult in a well-developed case. It should be remembered, however, that amyotrophic lateral sclerosis bears a certain superficial resemblance to myelitis of the cervical region with descending degeneration, and that it may be simulated by certain cases of disseminated sclerosis.[8] Upon its distinction from progressive muscular atrophy we have already dwelt. With respect to progressive bulbar paralysis, it may be doubted whether those cases where disease of the pyramidal tract is present can always be distinguished during life from those where it is not; but if so, it will be done by careful observation of the tendon reactions. Indeed if the assumption, suggested above, be justifiable, that disease of the motor tract may begin in any part of it, it is possible that many forms of progressive muscular atrophy, peripheral or central, of bulbar paralysis with or without atrophy of the limbs, and typical amyotrophic lateral sclerosis, may be expressions of one and the same proclivity to disease of the motor apparatus, differing only in its localisation, diffuseness, and intensity.[1]

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[1] 'Gazette hebdomadale,' 1867, p. 453; quoted also by Leyden; and Gombault 'Thèse de Paris,' 1877, p. 66.

[2] 'Arch. der Psych.' vol. viii. p. 295.

[3] "Brain," No. 32.

[4] Ziemssen's 'Medicin.' Later German writings say, however, this case was not amyoilophic lateral sclerosis. See Pick, 'Real-Encyclop. für gesammt. M.,' vol. xii. p. 643.

[5] 'Deutsch. Zeitschrift f. pract. Medic.' 1876, Nos. 29 and 30.

[6] In the case quoted later on.

[7] 'Arch. f. Psych.' vol. x.

[8] Déjérine, 'Revue de Médecine,' 1884. "Sclérose en plaques cérébro-spinale à forme de sclérose latérale amyotrophique."

It remains to discuss in more detail the morbid anatomy of the disease.

The broadest and most important feature thereof is this, that it affects solely or principally the motor tract, and may affect the whole length of it, from its origin in the cells of the cerebral cortex to its termination in the muscles.

Only the striated muscles are diseased, and principally those of the hands and arms, tongue, lower face and neck. The minute changes in them are somewhat various; there may be simple atrophy, diminution in size and number of the fibres without loss of striation, or there may be granular or fatty disintegration of the muscular substance with or without multiplication of the nuclei of the sarcolemma. The interstitial tissue may either be normal, or increased in amount, or infiltrated with fat. Mention must here be made of a remarkable case published by Barth.[2] In the muscles was found a condition corresponding with that described in pseudo-hypertrophic muscular paralysis. The interstitial tissue of the muscle was increased in amount and enormously infiltrated with fat; the fibres were in part healthy and in part degenerating. But in the cord lesions were found corresponding to those of amyotrophic lateral sclerosis, described clearly enough by the author and corroborated by Flechsig.[3] The disease had begun (in a patient aged 44) with progressive paralysis of the legs and then of the arms, accompanied by muscular twitching. There was wasting when first observed; but five months before death the calves and adductors of the thumbs began to enlarge, though without regaining strength.

In the nerve trunks and nerve roots it is to be noticed that wherever motor fibres run separately from sensory, the former only are diseased. Thus the anterior spinal roots (of the cervical region particularly), the hypoglossal roots and sometimes other of the cerebral motor nerves, look thinned and grey, while the posterior nerve roots are always healthy. The minute changes in the nerves are less striking than might have been expected; sometimes none can be made out, sometimes there is simple atrophy or degeneration of the nerve fibres.

The lesions within the cord constitute the most important and most constant feature of the disease. Of these we distinguish—*firstly*, a well-marked degeneration affecting those parts which are definitely known to be motor in function. Thus in the grey matter only the anterior cornua are diseased; the large multipolar cells undergo atrophy or pigmentary degeneration, and although the other elements may be affected too, there seems reason to think that the disease begins in the cells. The cells of Clarke's columns and of the intermedio-lateral tract are usually, but not always,[1] normal. Ordinarily the grey matter of the cervical region is most deeply diseased. Again in the white matter there is a well-marked degeneration of the pyramidal tracts; the crossed pyramidal tracts being always diseased, and frequently the direct pyramidal tracts also. Cases in which the latter remain apparently healthy may be explained on the supposition, that there has been a total decussation of the pyramids. The disease in the white matter has, so far, the distribution of bilateral descending degeneration, secondary to transverse lesion high in the cord or bilateral lesion of the cerebrum; but the absence of such lesion proves that this is not its true character. But *secondly*, even in distribution, there are differences between this disease and descending degeneration. The stress of it falls, it is true, upon the pyramidal tracts, but it is usual to find other parts of the white matter diseased as well. This, like the disease of the anterior cornua, is observed principally in the cervical region. Here though the area of degeneration as seen in transverse section is marked out sharply on its peripheral side, where it corresponds with the limits of the crossed pyramidal tract, it is not necessarily so elsewhere. Thus anteriorly there is no such well-defined limit, but the degeneration spreads, though becoming fainter, beyond the area of the pyramidal tract into the remainder of the lateral column towards the anterior cornua. Internally also it may spread, in some cases,[2] up to the margin of the grey matter. The anterior root zones may also be diseased. As we pass down the cord all these subordinate areas of degeneration disappear, the disease limiting itself more and more closely to the area of the crossed pyramidal tract, till in the lumbar region these tracts only are affected.

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[1] Kahler, 'Zeitschrift der Heilkunde,' 1884, 109.

[2] 'Arch. d. Heilkunde,' vol. xii. "Atrophia musculorum lipomatosa."

[3] 'Arch. d. Heilkunde,' vol. xix.

The meaning of these minor areas of degeneration is not altogether obvious. Perhaps that in the anterior root zone may be caused by irritation from the diseased anterior root fibres which pass through these parts, for the same thing has been observed in simple anterior cornual myelitis (infantile paralysis).[3] Whether the area in the lateral columns, anterior to the pyramidal tract, is also caused by irritation, or whether it be due to disease of some intraspinal tracts of fibres, the nature and course of which we do not yet know, must be regarded as uncertain. Flechsig, however, argues strongly that it is not caused by a diffuse myelitis; for were that the case, the adjacent cerebellar tract would be almost certainly involved; but this is never so.

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[1] Charcot, 'Leçons,' vol. ii. p. 423.

[2] Charcot, 'Leçons sur les Localisations,' &c., p. 161 note.

[3] See Moeli, 'Arch. f. Psych. vol. x.

We must note, before leaving the cord, that in some few cases[1] slight disease of the posterior columns has been found, but this is usually regarded as non-essential.

Passing upwards, the disease may be traced through the pyramids into the pons, being still limited to the fibres of the motor tract. Usually it ceases here; we shall quote some important exceptions to this presently. The cells of the motor nuclei are atrophied, notably the hypoglossal, sometimes also those of the vagus, spinal accessory and facial. In a case by Maier[2] the olivary bodies, both in their white and grey matter, were diseased; no other example of this is on record.

The distribution in the nervous centres is of more importance than the histology. For if, as has been stated, the distribution is practically coincident with that of the motor tracts, then we conclude (with Flechsig) that the disease originates in the nerve-elements. For it is only with respect to their nerve-function and development, and not with respect to their vascular supply or interstitial tissue, that these tracts are in any way differentiated from the surrounding parts. And on the other hand, as some authors maintain,[3] degeneration, whether parenchymatous or interstitial in origin, tends in the long run to assume the same histological characters; so that deduction from the microscopical characters can be made only in quite recent cases. Briefly stated, however, the changes in the white matter are as follows. In the areas of complete degeneration (pyramidal tracts) there is well-marked overgrowth of interstitial tissue, and wasting or absence of nerve-fibres. Leyden says that in his cases the axis cylinders were never hypertrophied, as in disseminated sclerosis. Charcot has noticed[4] that even in the most deeply affected parts of the pyramidal tract a few healthy nerve fibres may be found, whereas in secondary descending sclerosis they all degenerated together; he thinks this may indicate that fibres, other than motor, run in these tracts. Where less complete, the disease is indicated by a diffuse overstaining in carmine; the normal septa of connective tissue are thick and the atrophy of nerve fibres is far less marked. In addition we must notice the presence, sometimes in large numbers, of rounded granular, probably fatty bodies. These, which may be connected with the breaking up of the myeline sheaths, are most important, according to Charcot, for tracing out the disease into parts where actual sclerosis has not yet begun.

We shall conclude with a notice of some important cases in which the lesion has been traced up into the cerebral hemispheres.

In Maier's case referred to above, and in some by Leyden, it had been found to reach into the crura cerebri. The first case in which the hemispheres were affected is recorded by Kahler and Pick.[1] This was a female, age 44. She had had twitchings of the arms, then weakness and wasting of them, then contractures. Simultaneously, weakness of the legs. Afterwards, affection of speech and swallowing. When first seen, the legs were paralysed, stiff and extended, their muscles not wasted; there was ankle-clonus and excess of tendon-reaction. The upper limbs were flexed, the hands and forearms wasted. The face was lax and expressionless, its lower part almost paralysed, slight ptosis. Tongue wasted and tremulous, speech almost unintelligible; soft palate motionless. Muscles of head and neck weak. No fibrillary twitchings. There was reduction of faradic contractility in the wasted muscles; to the galvanic current no distinct R D; K S Z > A S Z everywhere, but in some muscles K O Z > A O Z. Duration short, eight months in all; death from pneumonia, food having passed into the air-passages. Post-mortem, degeneration was found of both the crossed pyramidal tracts, the characters thereof being such as to lead the authors to think that it was parenchymatous in origin. This was traceable into the pyramids, pons and crura cerebri. The grey matter of the anterior cornua was diseased. In addition, there was atrophy of the cerebral convolutions, especially marked in the neighbourhood of the central and præcentral sulci.

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[1] As in Leyden's case mentioned above; and the two quoted from Charcot and Marie at the end of this paper.

[2] See Virchow's 'Archiv,' vol. lxi.

[3] Déjérine, 'Arch. de Physiologie,' Nov. 15, 1884.

[4] 'Archives de Neurologie,' No. xxix. p. 173

In this case, the connection of the cerebral atrophy with the disease of the motor tract lower down was more or less a matter of inference; but in the next, the disease was actually traced up through the corona radiata to the cortex cerebri. The case is given by Kojewnikoff.[2] A man, aged 29, was seized with tremor in the right foot, followed by loss of control over it, and occasionally involuntary movements of extension in it. In a year, similar affection of the left foot, difficulty in movements of trunk, and in another year, of the hands. When first seen, there was difficulty of walking, stiffness of the limbs, exaggerated tendon reactions; electro-contractility of muscles normal. When seen again after a year's interval, there was further loss of power, and a spastic state of the legs; atrophy of the thenar and hypothenar muscles, with some loss of electro-contractility. There were no bulbar phenomena. Phthisis had now developed in the lungs, and the patient died about 3½ years after his first symptoms. Post-mortem, there was sclerosis of the crossed pyramidal tracts in their whole lengths, which in the cervical region spread forwards into the rest of the lateral column, and was here accompanied by alterations of the anterior cornual cells. The sclerosis extended into the pyramids. Numerous granular bodies were found (as also in Kahler and Pick's case) in the sclerosed tracts. But by the presence of these bodies the disease could be traced further upwards, viz. into the pons, into both cerebral peduncles (middle third of each), and thence into the hemispheres, where it spread through the internal capsules (the third quarter, from before backwards, of the posterior half of each capsule), and through the corona radiata, wherein the track of the disease spread upwards, radiating like a fan, to the upper part of each ascending frontal convolution. The grey substance of the convolutions, was, however normal.

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[1] Prager, 'Vierteljahrschrift,' &c., 1879. Bd. 142, p. 72.
[2] 'Archives de Neurologie,' No. 18.

The last advance has been made by Charcot and Marie, who have been able to demonstrate in two cases[1] not only the presence of granular bodies in the centrum ovale, but the disappearance of the large pyramidal cells in certain regions of the cerebral cortex. The first was the case of a woman, aged 60. The course was rapid, proving fatal within a year; the clinical phenomena are thus summarised by the authors. Onset with stiffness of left leg. Six months afterwards, paresis of the right arm, slight difficulty of speech. Two months later, atrophy of the thenar eminence (right), increase of the difficulty in speech. Next, complete paralysis, with spastic symptoms, which disappeared later on, increase of the atrophy in the hands, troubles of deglutition, respiration and circulation. Death in a year's time.

An electrical examination by Dr. Vigouroux showed, in the upper limbs, that there was considerable diminution of both faradic and galvanic contractility; no distinct reaction of degeneration, but A S Z = K S Z in several muscles.

The second case (also a woman of 60) lasted two years. Onset, with difficulty of speech and of deglutition. Four months later an apoplectic attack, followed by increase of bulbar symptoms. Paresis, then atrophy of the upper limbs, paralysis of the lower limbs with spastic phenomena.

The bulbar symptoms in this second case were very marked, and existed from the commencement; yet, contrary to what might have been expected, the fatal issue came less soon than in the others. The morbid anatomy of these two cases did not differ materially, and may be summarised as follows. In the *spinal cord* there was sclerosis of both crossed and direct pyramidal tracts, which in the cervical region extended over a considerable part of the remaining antero-lateral column; there were the usual changes in the grey matter. In the diseased parts of the cord there were a large number of granular bodies. There were indications of sclerosis also in the posterior columns, but no granular bodies. The authors are inclined to suppose, that the disease was here different in nature, and perhaps merely coincident with the degeneration of the motor tract.

There were granular bodies in the pyramidal tract within the crura cerebri, and within the pons, so far as examined.

Within the *hemispheres* these bodies were found in the internal capsule, occupying a smaller area than that of the pyramidal tract, though comprised apparently within it. Just as in Kojewnikoff's case, they spread up through the corona radiata, in a fan-like fashion, following the direction of the white fibres, to the cortex of the ascending frontal and parietal convolutions, chiefly at their upper portions at the level of the paracentral lobules. In the grey matter here were no granular bodies; but it was found, on the other hand, that the large pyramidal cells, commonly supposed to be motor in function, were absent or diminished in number. Those that remained were altered, their bodies less voluminous, their angles less marked than normally.

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[1] 'Archives de Neurologie,' Nos. 28 and 29.

Much interest attaches to the morbid anatomy of these latest cases, as showing, that in the cerebrum as in the cord the disease affects the elements of the motor tract; and secondly, that it may affect any level of the motor tract, the cells of the cortex cerebri the white substance of brain or cord, the anterior cornual cells of the cord, the nerve trunks, or the muscles.

RECENT PAPERS ON CONCUSSION OF THE SPINAL CORD.

BY HERBERT W. PAGE.

It is almost impossible in the present day to write on concussion of the spinal cord without reference to the subject of railway injuries; but whether the connection be illicit or legitimate, there can be no doubt that, by itself, concussion of the cord is of intrinsic interest to surgeons and neurologists, because of the uncertainty there always is in deciding whether in company with, or, what is of greater importance, without, an injury to the spine the cord has really been injured by concussion alone, and because of the question, whether late secondary degenerative changes in the cord can have this origin, even when there are no immediate symptoms of injury. It is now pretty well agreed, we think, that true concussion of the cord occurs with extreme rarity, and although a considerable number of cases have been recorded as examples of concussion-lesion, it is nevertheless certain that in many of them sufficient attention has not been paid to the possibility of the cord having been injured in some less obscure manner. If we put aside certain cases which may fairly be excluded—those for example, in which there has been fracture-dislocation of the spine with direct contusion of the cord—and look for examples of true concussion amongst those where the cord has been injured without obvious structural damage to the spinal column, then we ought also to exclude those cases in which the history of the accident makes it highly probable that the cord has been locally damaged by severe and sudden bend. This point has been dealt with by the writer in his work on 'Injuries of the Spine,' &c., which has been noticed in previous numbers of 'Brain,' and cases of the kind are there recorded which in his judgment cannot fairly be regarded as instances of true concussion. To that work nearly all recent writers on "Concussion of the Spinal Cord" make reference, and MM. Duménil and Petel in the series of papers before us [1] deal at considerable length with the objections which we have raised, not, as they seem to think, against the very existence of concussion of the cord, but against the too ready assumption of concussion as the cause of cord injury in particular cases, and therefore against the supposed commonness of this kind of injury, especially in a peculiar form of accident. They themselves record a case which is to them a clear example of concussion-injury, a case, moreover, in which, as they believe, a true system-degeneration had origin in concussion pure and simple. It is a case of much interest and importance; but, before examining it, we would point out that nowhere have we said that concussion of the cord is an impossibility and has never occurred, and cases are even recorded in our work which, as far as can be told, are explicable only on that hypothesis, notably the well-known case of Dr. Bastian's and a few others. Nor have we denied that the famous railway-accident case of Mr. Gore *may* be a case of concussion-lesion; but we have asserted, and do again assert, that there is a gap in the history of that case which renders it a not unequivocal example of such an injury, that it is a case, therefore, on which no lasting doctrine should be based, especially when it is remembered that the post-mortem appearances were singularly like those of posterior sclerosis as found in tabes dorsalis (locomotor ataxy). It is difficult to conceive how an effect so general as concussion can fall only on limited tracts of the cord, and to explain such cases it has to be assumed that a "predisposition" to disease allows concussion to exert a morbid influence.

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[1] 'Archives de Neurologie,' Nos. 25, 26, and 27 (1885).

The consideration of a large number of recorded cases, and the experience of surgeons, alike show, we believe, that true concussion of the spinal cord from the ordinary accidents of everyday life is amongst the very rarest of injuries, and that being so, it is at any rate improbable that concussion should be a common result of any one peculiar form of accident, such as a railway collision. The question, however, is far too important to be decided by a mere improbability, and the writer therefore investigated the whole matter of the spinal injuries of railway accidents, bringing to bear upon that investigation the personal experience of several hundreds of cases, many of them being inquired into long after the accidents, and calling to his aid the experience of others by the method known as "collective investigation." The result of that inquiry was to show, that not only are injuries to the *spinal column* and the soft structures outside it extremely common in railway accidents, but also that the "railway spine," as it has been called, is not, and has no resemblance to, concussion of the spinal cord—a conclusion which neither shuts out, nor indicates a disbelief in, the possibility of there being such a thing as concussion of the cord, or that the cord *may* be injured by concussion even in railway collisions. Not only as the result of any and every form of accident, but also as the result of the special form of accident met with in railway collisions, concussion of the spinal cord is amongst the very rarest of injuries, using the word "concussion" to signify an effect upon the cord of the same nature as the effect upon the brain from a concussion-blow, the results of which may vary from simple shake of the brain-mass to actual contusion or laceration of its substance. The case, however, recorded by MM. Duménil and Petel, is not a case of railway injury. It is given at great length, and it is impossible to do more than refer to its salient features here.

A man, aged 60, fell from a height of about five feet vertically upon his head. There was complete loss of consciousness for about an hour, and when he came to himself there were all the evidences of a severe injury to the cord in the lower cervical region, complete paralysis of the lower limbs, almost but not quite complete paralysis of the upper, that of *the right arm being more marked than the left*, such movements as were possible being performed by the shoulder muscles, and diaphragmatic breathing. For a day there was abolition of sensation below the umbilicus, loss of control over the sphincters, &c. Sensation gradually, but never completely, returned in an irregular manner, and was replaced by hyperæsthesia in various parts, and shooting-pains in the limbs. At a later period there was marked rigidity in the lower limbs, with hyperæsthesia here and there, and variable attacks of pain in the extremities. There was pain on moving the neck, mostly at the level of the sixth cervical vertebra, but no discoverable deformity. The case is recorded with minutest detail from day to day, and seems to be divided into two periods: the first, of forty-seven days without fever, and a gradual, though very slight, tendency to restoration of function; followed by a second period of five months, which was ushered in with shivering, and was marked by various disturbances of motion and sensation, bed-sores, purulent urine, seemingly indicative of a more active myelitis, and then the patient died.

Post-mortem, both spine and membranes were perfectly healthy, but we read that "à la partie supérieure de la région dorsale, la coupe présente une dépression au niveau de la corne antérieure droite," and that, microscopically, there was found degeneration; commencing sclerosis of the crossed pyramidal tracts, slight in the cervical region, apparently less marked in the lumbar than in the dorsal, but in the dorsal more so than in the cervical region; and also "granular degeneration of the motor cells of the grey substance, especially in the dorsal region." From all the circumstances of this patient's history it is perfectly clear that his illness and death, and the post-mortem appearances, were due to the accident, but it is by no means so clear that the original lesion was caused by concussion. Nay, this seems to be one of those very cases where sufficient allowance has not been made for the possibility, and here the exceeding probability, of the cord having been locally damaged by severe and sudden bend. The man fell vertically on his head, and was momentarily pivoted, so to say, with his head downmost, his body uppermost, and his neck intervening; and, with the tremendous leverage of the body-weight, his spine and spinal cord were inordinately bent somewhere in the cervical region. The spine itself—with its great flexibility in this part—does not give way; but the cord yields under the sudden strain, yields most in its anterior parts, if we assume that the bending was backwards rather than forwards. He has pain about the region of the sixth cervical, and somewhere about that level the cord is obviously severely damaged, for he is almost completely paralysed, and diaphragmatic breathing alone remains. The symptoms, moreover, seem to show that this lesion could not have been a concussion-lesion, and that the whole cord, at any rate, was not affected by concussion; for let it be noted that the man was unconscious for an hour, and there therefore was concussion of the brain. Concussion, it is assumed, next fell on the cord about the level of the sixth cervical, and involved the whole of the cord beyond that point. But why should, how indeed could, that portion of the cord which intervened between the brain and the sixth cervical have been excluded from the force or effect of the concussion, transmitted, as it must have been, from the vertex of the head? That it was not involved in the concussion is pretty evident from the fact, that diaphragmatic breathing remained, the cord origin of the phrenics being quite unaffected, being indeed above the level of the gross lesion, which had been determined, it seems to us, by bend at a particular point, and not by concussion at all. It is suggested, however, that the sclerotic degeneration was also due to, and was a later consequence of, concussion of the cord. It is true that no special evidences of injury could be found at any one point of the cord, 169 days, that is, after the accident, and that the authors therefore, do not attach much importance to the "depression" previously named; but this degeneration looks very like a true secondary degeneration spreading from some point of lesion in system-tracts, having a chief tendency to spread downwards, and getting less marked the further it was removed from that point. In other words, the case was probably one of secondary degeneration of the crossed pyramidal tracts after a local lesion of the cord, a thing more likely to have occurred than for those tracts to have been specially picked out and exclusively affected by concussion. It was no unusual circumstance that at a period remote from the accident the patient should have been attacked with a somewhat diffused myelitis, but there is no evidence that that was in any way the result of concussion. One cannot help thinking that the interpretation here put upon the case is more consonant with the facts than is that which attributes the phenomena, immediate and secondary, to concussion and concussion-lesion of the spinal cord. Apart, however, altogether from this case, the paper is of much value, for the authors endeavour to elucidate the symptomatology of concussion of the cord. They especially point out the differences which they believe exist between concussion symptoms and those due to intraspinal hæmorrhage, though we doubt whether it is warrantable to draw any very precise conclusions from their own case. And here we think they have misunderstood the contention of the writer, who has attributed some of the recorded cases of supposed concussion to hæmorrhage. In the work referred to we quoted a case of Mayo's, where a man had a violent blow on the lower part of the back and died in four hours, blood being formed in the spinal canal, with perfect integrity of the spine and spinal cord. Evidently, say the authors, it was not extravasation of blood which killed the patient in four hours. Clearly not; the history indeed throws no light on the actual cause of death, which nevertheless *may* have been due to shock from concussion of the cord, an explanation which at the very best is simply hypothetical. The history of that case, however, suggested a probable explanation of a case of Sir A. Cooper's in which there was no necropsy, and also showed that even a violent blow such as the patient received direct upon the spine, caused no structural injury to the spinal cord, an evidence of the secure protection of that organ within the spinal canal.

The following are the conclusions at which MM. Duménil and Petel arrive. (1.) That there is such a thing as concussion of the spinal cord; and (2), that it is the cause of consecutive inflammatory mischief which (3) may assume the form of a "systematic" myelitis; and (4) that concussion of the cord may exist as a latent condition, and reveal itself only by secondary troubles, which may take the forms of a simple transient congestion, or of an incurable sclerosis. Notwithstanding the authors' own case and that of Mr. Gore, conclusion 3 yet lacks, we believe, the foundation of an absolutely unequivocal case, for, as we pointed out in a previous number of 'BRAIN,'[1] the cases of tabes dorsalis of supposed traumatic origin which were collected from various sources by M. Petel, are surrounded with doubt. It seems to us, moreover, that conclusion 4 ought only to be based on definite examples, for several cases which fall in this category, amongst them being the perennial case of the Count de Lordat, have been in all probability cases of local spinal injury with few or no symptoms at first, but in which there was gradually set up a local pachymeningitis, followed by later degeneration in the cord.

A case recorded by Professor Verriest[1] is briefly as follows. A heavy mass of coal fell on the lumbar and sacral regions of a man aged thirty-four. He was picked up insensible, and was found palsied in all his limbs. Great pain in the lumbar region extended up the whole column. Next day he vomited a large quantity of blood, and in three days power began to return in the right arm, then in the left, and lastly in the legs. He gradually improved, but his muscular power remained very feeble. The reflexes were but little altered, and sensibility was only slightly lowered. He had pain on pressure of the spine and on movements of the trunk. He was then lost sight of for twenty months, when he was found in a most feeble state; wasted, easily fatigued, his reflexes exaggerated and sexual power gone; subject to vomiting and dyspnœa; with a slow pulse, his intellect feeble, and double optic neuritis. This case is regarded by the author as one where concussion was the starting-point of an insidious meningitis and myelitis, and we cannot help thinking that it is much more suggestive of concussion than the case of MM. Dumenil and Petel, difficult though it be to account for the insensibility on any mere concussion theory. The double optic neuritis points in all probability to a meningitis which had spread up to the base of the brain, while all the symptoms may be accounted for by a widely diffused spinal meningitis with irritation or involvement of nerve-elements. That being so, it is not altogether improbable that the real cause of the mischief was originally an intraspinal hæmorrhage, and it is at any rate clear that there was no intervening or latent period, but a steady progressive degeneration started by the injury sustained.

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[1] Vol. II. p. 440

[2] 'Revue Médicale de Louvain,' No. 12, 1885.

Dr. Dana's paper "on Spinal Concussion and the nature of its Sequelæ," and the discussion which followed it,[1] dealt more largely with concussion of the cord in its relation to railway injuries. A large number of cases of supposed concussion are collected by Dr. Dana, whose concise and impartial remarks on each of them will well repay perusal. In the debate which followed, Dr. Hammond referred to a case which he "could not conceive as anything else than a concussion," where a woman had fallen, doubled-up, thirty-five feet on her buttocks. There was no fracture, and when seen three months afterwards by him, she "was still suffering from paraplegia, and incontinence of urine and paralysis. Hypochondriasis and hysteria had also developed to an extreme degree. I do not think there was any hæmorrhage in her case, because I do not think hæmorrhages of the spinal cord are recovered from. She suffered not only from motor troubles, but from sensory disturbances likewise; also from atrophy of her limbs, showing that the injury involved the whole segment of the cord, both the sensory and motor parts. Yet she has recovered, and I do not believe, therefore, that in her case there was any destruction of the cord. I cannot conceive that there could have been anything else than a concussion." One would have thought that the scientific imagination of Dr. Hammond might have conceived something else, say, for example, "spinal anæmia," or "spinal hyperæmia"; but we cannot gather from his remarks what is exactly meant by "concussion" in this somewhat obscure, or inadequately reported case—whether "concussion" is used to indicate cause or effect.

Dr. Weber made the observation, that while he would not say there was no such thing as concussion of the spinal cord, "he believed that a good many cases which are considered as such are not, in spite of the symptoms pointing that way, dependent on concussion of the spine, but upon concussion of the brain"—a criticism which applies with becoming force to many of the cases referred to by the speakers in this very debate. These are the chief of Dr. Dana's conclusions: 1. That the term "spinal concussion" is a misleading and often incorrect one, and that the symptoms which are usually associated with that name are really symptoms of traumatic neurasthenia—a term which covers a well-recognised symptom-complex—hysteria and hypochondriasis, associated more or less with symptoms of injury to the vertebral muscles and ligaments, and to the spinal nerves; that, in other words, spinal concussion is mental shock and physical bruising . . . . 5. That the prognosis of railway or traumatic neurasthenia and hysteria is very good, so far as steady improvement is concerned, not so good as regards complete recovery. 6. That concussion of the spinal *cord* alone, followed by temporary loss of function, or by myelitis, does occur in rare instances. 7. That, in the predisposed at least, injuries and jars may set up chronic myelitis, without there being a lesion of the spinal column."

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[1] 'New York Medical Record,' Dec. 6, 1884.

What then is the conclusion of the whole matter? Erb has given it in 'Ziemssen's Cyclopædia.'[1] "We do not yet know what changes, if any, constitute the basis of the concussion proper. . . The diagnosis is in many cases so uncertain, and the want of satisfactory evidence from autopsies so great, that the history of the disease is still surrounded by darkness. . . It is therefore rather rash to entertain a decided opinion regarding the *proper nature of concussion of the cord.* It seems to be certain that the anatomical report is a negative one. The most common view, therefore, is that which supposes only molecular changes in the finer nerve-elements to have occurred, giving rise either to an immediate and complete functional paralysis of the latter, or forming the commencement of further disturbances of nutrition, which at a later time may result in degenerative inflammation. . . . Much remains to be done in the more correct estimate and clearer definition of cases; the first thing consists in collecting accurate reports of cases, avoiding, more carefully than has hitherto been done, the intermixture of other sorts of lesions." These words, "avoiding the intermixture of other sorts of lesions," should be borne in mind by every one who has the opportunity of observing, or who attempts to describe, a case of that seemingly rarest and most elusive of injuries, concussion of the spinal cord.

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[1] Vol. xiii. p. 344.

ON CEREBRAL INFANTILE PARALYSIS, SPASTIC CEREBRAL HEMIPLEGIA (HEINE), POLIENCEPHALITIS ACUTA (STRÜMPELL).

BY H. HANDFORD, M.D., M.R.C.P.,

Physician to the Nottingham General Hospital.

1. Strümpell: 'Deutsch. Med. Woch.,' No. 44, 1884.
2. Ranke: 'Münch. Med. Woch.,' No. 17, 1886.
3. Langenbeck: 'Deutsche Klinik,' 1863, S. 51.
4. Sander: 'Centralblatt für die Med. Wissensch.,' No. 15, 1875.

In 1884, Professor Strümpell of Leipzig wrote an article on acute encephalitis in children, or, as he called it, polienccphalitis acuta, or cerebral paralysis. Though the existence of such cases has been recognised clinically, and they are found not to be rare when carefully sought, yet hitherto no very satisfactory account has found its way into the text books.

Acute encephalitis in children resembles very closely infantile paralysis of spinal origin. Like poliomyelitis, it attacks usually healthy children. The limits of age in Strümpell's cases were from four weeks to six years. No special exciting cause could be traced in the great majority of cases; injury to the head either during birth or subsequently seemed to start the mischief in some cases.

The attack begins usually quite suddenly with sickness, convulsions and feverishness; of these symptoms, convulsions are the most constant, and when they have passed off, the resulting paralysis is generally noticed by the parents. The hemiplegia is seldom complete, and most children learn to walk again after a time, though imperfectly. The arm is more affected than the leg, but the paralysis of the arm is not so complete as it often is in poliomyelitis. The facial muscles are seldom involved. Besides hemiplegia, crural or brachial monoplegia may result, and sometimes merely a kind of ataxia affecting all movements. The affected limbs are usually smaller than those on the sound side, but there is no true atrophy comparable to that of poliomyelitis, and consequently no reaction of degeneration. The deep reflexes are exaggerated. Of special interest are certain signs of motor irritation, because they may with great probability be referred to the cerebral cortex as their seat. Some cases become epileptic, and then the convulsions commence almost without exception on the paralysed side, and are sometimes limited to it, in such cases passing off without loss of consciousness. A more frequent result than epilepsy is the condition of irregular movements, limited to the paralysed extremity and frequently to the hand, called athetosis. The intellectual faculties may remain quite unaffected. Not unfrequently, however, they show some disturbance. The speech is sometimes impaired in right-sided paralysis. Sensation on the paralysed side is little, if at all, interfered with.

The supposition, that the cerebral cortex is the seat of disease in this form of hemiplegia, is supported not only by the clinical picture, the distribution of the paralysis, the epileptic attacks, and the athetosis, &c., but also by the results of a considerable number of necropsies. Changes evidently of inflammatory origin have been found in the cortex of the motor area, closely resembling the cicatrices and contractions in the anterior horns in poliomyelitis. Naturally, in making a diagnosis, one must not forget that there are other causes of hemiplegia in children than the above.

Professor Ranke's observations agree in the main with Strümpell's, but he attaches much less importance to the occurrence of fever at the outset, in fact in the majority of the cases he describes, no history of a feverish attack could be obtained.

In the ten months ending Feb. 1886, Professor Ranke met with twelve cases of infantile spinal paralysis, and eleven of cerebral paralysis; these last agreed in all their main features with the descriptions given by Heine and Strümpell. Of the eleven cases seven were female and four male; eight were affected with hemiplegia, three with monoplegia (one brachial and two crural). In all the illness commenced before the end of the fourth year, and in three the parents thought the affection was congenital. In the hemiplegic form, the arm was always more affected than the leg. There was considerable wasting of the paralysed limbs, chiefly affecting the muscles and bones, the fat and subcutaneous tissue not being much diminished. The paralysed limbs were generally shorter than those on the sound side. In seven of the hemiplegic cases, athetosis occurred ; in one involving the whole arm and leg on the paralysed side, and in the rest limited to the fingers and wrist, except that in four the toes were similarly affected. In one hemiplegic case, and one of brachial monoplegia, there was no trace of athetosis.

Disturbances of the intellectual faculties were observed in three of the hemiplegic cases. One boy, in whom the paralysis came on shortly after birth, became epileptic when four years old, the convulsions always beginning on the paralysed side.

The paralysed limbs were not flaccid, as in poliomyelitis, but in all cases there was muscular tension and slight rigidity, leading in one case to marked contraction. The tendon-reflexes were present in all the cases, but no exaggerated reaction was detected, as stated by Strümpell. No diminution of sensation on the paralysed side could be detected. The electrical examination was carried out with the greatest care, the galvanic current was measured in milliampères, and the faradic by means of the Du Bois Reymond sledge. In no case was the reaction of degeneration present, and no definite and constant difference in the excitability of nerve and muscle on the two sides could be detected in any of the cases. Infantile cerebral paralysis is not generally dangerous to life, and as none of Professor Ranke's eleven cases terminated fatally, no pathological results could be given. Strümpell's experiences have been referred to above. Langenbeck and Sander also give pathological details. Professor Kundrat, in his monograph "Die Porencephalie," Graz, 1884, has collected forty-one cases. He describes marked changes in the cerebral cortex, and likewise in the deeper parts. The exact pathological anatomy of infantile cerebral paralysis is not yet definitely settled.

Four well-marked cases that have survived to adult life have recently come under my care. In one of them a careful electrical examination was carried out, the galvanic current was measured in milliampères, and certainly no trace of reaction of degeneration was present. The reactions were normal, and no difference on the two sides could be detected. The patellar tendon-reflex on the affected side was exaggerated.

Professor Ranke gives the following table for the differential diagnosis of infantile spinal and cerebral paralysis.

INFANTILE SPINAL PARALYSIS.	INFANTILE CEREBRAL PARALYSIS.
1. *Mode of Onset* (initial stage.)	
Fever, convulsions, sometimes with loss of consciousness. Any of these symptoms may be absent, or the whole stage may be slightly marked.	The same symptoms; but a definite onset seems more frequently to be absent.
2. *Age.*	
The paralysis begins from the 1st to the 4th year, seldom later.	As in spinal paralysis, but some cases seem to have been noticed from birth.
3. *Form of Paralysis.*	
Monoplegia most common. Then paraplegia. Hemiplegia very rare.	Hemiplegia most common; then monoplegia. Paraplegia very rare.
4. *Nature of the Paralysis.*	
Flaccid: contractions form only after long duration of paralysis.	Spastic, slight condition of muscular tension present from the beginning.
5. *Electrical reactions of muscles and nerves.*	
Excitability to faradism greatly diminished. Reaction of degeneration present.	Excitability to faradism preserved. No trace of reaction of degeneration.
6. *Sensation.*	
Unaltered.	Scarcely perceptible alteration.
7. *Tendon reflexes in paralysed limbs.*	
Absent.	Present, often exaggerated.

8. *Temperature of paralysed limbs.*

Markedly diminished.	Slightly diminished.

9. *Colour of paralysed limbs.*

More or less livid.	Unaltered.

10. *Retardation of growth.*

Strongly marked.	Generally strongly marked, and then chiefly in the upper extremities.

11. *Signs of motor irritation in the paralysed limbs.*

Absent.	Athetosis common, sometimes epilepsy, the convulsions beginning on the paralysed side.

12. *Disturbance of intellectual faculties.*

Absent.	Common.

13. *Disturbances in the functions of bladder or rectum.*

Absent.	Occasional and slight.

14. *Contractions as results of paralysis.*

Talipes Varus „ Valgus „ Calcaneus „ Equinus and combinations of these; and frequently also spinal curvatures.	No other deformity than Spastic talipes equinus, and contraction of the knee, and occasionally also adduction of the thigh. According to Heine spinal curvatures are never met with.

Reviews and Notices of Books.

La Psychologie du Raisonnement, recherches expérimentales par l'hypnotisme. Par ALFRED BINET, pp. 171. Paris: Félix Alcan.

THE title of this volume hardly indicates its full scope. It is not merely a study of the reasoning process; it includes a pretty full investigation of the act of perception, and makes important contributions to the theory of images. The aim of the writer may perhaps be defined as the explication of the complex mental operations of reasoning by a preliminary investigation of more elementary processes. More particularly the author makes use of the recent researches in the field of hypnotic hallucination in order to throw light on the normal processes of inference. The interest and value of the work depend, to a large extent, on the writer's intimate knowledge of the phenomena of images, both in normal and in abnormal states, as elucidated by recent experiments. To this it may be added that it is the production of a member of the new school of physiological psychology which is doing so much useful work in France just now. Like his previous work on Hallucinations, it appeals to all those who are interested in mental phenomena in their connection with the general functions of the nervous system.

The volume opens with a general account of perception as a process in which an impression of sense is completed "by an escort of images." On this follows a highly interesting chapter on images. Mr. Binet has a good deal to say about individual differences in respect of imaging. He distinguishes between "indifferents" those who can image sights, sounds, and movements, with equal readiness, and "visuels," "auditifs," and "moteurs," that is to say, persons who represent vividly and predominantly one of these classes of sensation only. By aid of this distinction he seeks to explain the discrepancy between the accounts of verbal representation given by M. Stricker and his critics. M. Stricker, who in representing words is distinctly conscious of incipient articulate movements, is, according to our author, a "moteur" of a very pronounced type, whereas in most cases words are recalled partly and mainly as auditory sensations or sounds. The writer then proceeds to discuss the physiological seat of images, and adds to evidence in favour of assigning to sensations and the corresponding images the same nervous centres a number of facts drawn from visual disturbances, and more particularly achromatopsy, occurring in hysterical patients. Such patients have been hypnotised with a view to note the effects of achromatism on the resulting hallucinations. Among the results of these experiments it was ascertained that the loss of sensibility to a particular colour precludes a hallucination or image of the same colour. Again, it was found that the excitation of a visual hallucination in a hypnotised subject modifies the sensibility of the eye, and that hallucinations of colour evoke complementary spectra, just as actual chromatic sensations evoke them. All these facts, says our author, " show the close analogy of the sensation, the hallucination and the image; one may conclude from them that whether one has the sensation of red, or the recollection of red, or sees red in a hallucination, it is always the same cell that vibrates." The writer then further enforces his conclusion by a reference to some researches carried out by M. Parinaud and himself on after-images. These he rightly regards as a transition from sensations to images of memory. In these experiments it was found that a negative or complementary after-image could be transferred from the eye that actually received the impression of the original colour, to the other eye which had been precluded from seeing it. This fact, if well-established, undoubtedly tells in favour of the exclusively central character of the phenomena. But in this case it is clear the very facts of negative after-images require another kind of explanation than that usually given, e.g. by Helmholtz and Hering. And it is not easy to see, at a first glance, how these complementary images are explicable as concomitants of compensatory cerebral actions. M. Binet does not attempt any such explanation. The whole subject would thus appear to require reconsideration.

After this study of images M. Binet returns to the subject of perception, and shows how the percept results from the combination of two elements, a sensory impression and " cerebral image exteriorised." Here, again, good use is made of the phenomena of hallucination; and, as in M. Taine's well-known work " On Intelligence," the percept and the hallucination are regarded as having the same structure. Among other interesting points dealt with here is the effect produced in a hypnotised person known as " systematised anæsthesia," and which consists in an induced blindness to a particular person or other object. It is found that as the anæsthesia disappears the subject passes through gradations of perception, cognition of the presence of a person preceding individual recognition. This analysis of perception leads on to an assimilation of it to a process of reasoning. Both, says M. Binet, are forms of indirect or mediate knowledge, both presuppose pre-existing mental states, and both proceed by the recognition of similarity between the new experience and the old. The identity of the process is further shown by an analysis of the reasoning operation as formulated in the syllogism, in which the author derives much aid from Mr. Spencer's theory of reasoning.

In a subsequent chapter M. Binet examines at considerable length the mechanism of reasoning. Here he seeks to define with precision the law according to which similar mental states fuse one with another, and throws new light on the limits of the local discrimination of tactile sensations as established by Weber's experiments. It is shown that in all perception and reasoning alike the first step is the assimilation of a present impression or fact to a previous experience or group of experiences, and that both in perceiving and in reasoning this assimilation may be automatic or unconscious. The next step in the process is the suggestion of some other fact associated by contiguity with the consciously or unconsciously revived impression. And the third and final step is the association of this with the new or present experience. These three steps are clearly involved in syllogistic reasoning, since, indeed, they correspond to the two premises and the conclusion thus:—

> This is a crystal.
> All crystals have a plane of cleavage.
> This has a plane of cleavage.

And M. Binet has shown much ingenuity in assimilating to the same fundamental type the process of perception itself.

Enough has been said to illustrate the penetration and skill which the author has brought to bear on his problem. His solution is not altogether, as he himself fully admits, an original one. Helmholtz, Lewes, and others have insisted on the fundamental identity of the acts of perception and reasoning. But M. Binet has carried the work of his predecessors one stage further, profiting by newer experimental inquiries. If one misses anything in the exposition, it is an attempt to differentiate the normal processes of perception and inference from those abnormal processes to which reference is so frequently made. To point out the conditions of sound knowledge is, no doubt, rather the business of logic than of psychology. Nevertheless there is a psychological difference involved, and it might have been well, perhaps, to try to define this with some precision.

JAMES SULLY.

On the Structure, Distribution and Function of the Nerves which innervate the Visceral and Vascular Systems. By W. H. GASKELL, M.D. F.R.S., 'Journal of Physiology,' Jan. 1886.

THE ordinary ramus communicans of the sympathetic consists of a white and grey root; but the white root is present from the second dorsal to the second lumbar only; the further down that one proceeds, the fewer are the white fibres. The white root consists of some fibres which go into the anterior and posterior roots, and may be seen as very small medullated fibres in the cross-sections of the roots, and some fibres which proceed outwards, and send branches to the lateral or vertebral ganglia, and branches which pass the lateral ganglia; the upper of these go to the cervical sympathetic, the lower to the splanchnics. The grey root sends some fibres inwards, which are lost in the connective tissue about the vertebra and dura mater, chiefly about the posterior root, and some fibres which go out to the lateral ganglia. In the second and third sacral rami communicantes small medullated fibres again appear, forming the main part of the nervi erigentes, which go to the hypogastric plexus without communicating with the lateral ganglia; thence some pass up to the inferior mesenteric plexus, and some downwards to the bladder, rectum and organs of generation. It is proposed to call the nervi erigentes the pelvic splanchnics, the upward thoracic fine white medullated flow the cervical splanchnics, and the downward thoracic flow the abdominal splanchnics. Small medullated fibres go from the cord and medulla to the spinal accessory; the branch to the cervical plexus, sterno-mastoid and trapezius takes all the large white fibre part; that to the gang. jug. vagi takes the small white medullated fibres. The glosso-pharyngeal, petrosals, and chorda tympani also contain small white medullated fibres, so that there is a cervico-cranial as well as a thoracic and sacral overflow of fine white medullated fibres.

The vaso-motor nerves for all parts of the body come out as fine medullated fibres in the anterior roots of the spinal nerves, from the second thoracic to the second lumbar, inclusive; they go to the lateral ganglia, where they become non-medullated, and thence to their destination either after joining other ganglia or not; hence the lateral ganglia should be called the vaso-motor ganglia, and all the evidence which has been hitherto brought forward that vaso-motor nerves come out from other anterior spinal nerves is worthless.

The circular fibres of the intestine, stomach and œsophagus are supplied by the vagus, which at its entrance to the diaphragm consists of non-medullated fibres which make their appearance just below the gang. trunci vagi. All the medullated fibres below this pass away in other branches (*e.g.* cardiac, pulmonary, laryngeal), hence the alimentary vagal branches lose their medulla in the gang. trunci vagi. In alligators and crocodiles this ganglion is at the upper part of the thorax, and is hence easily placed for experiment. After the vagi are cut, the animals die of starvation, as shown by signs of inanition and the disappearance of fat. If the vagus was cut above the ganglion, its inhibitory power over the heart disappeared soon, even if the stimulus is applied below the ganglion, hence the cardio-inhibitory nerves do not end in the cells of the ganglion. Stimulation of different parts of the vagus showed that the motor nerves of the cervical œsophagus pass directly to it, probably by the large conjoint vagus and glosso-pharyngeal ganglion and pharyngo-laryngeal nerve, whilst those for the thoracic portion and stomach and intestines pass through the gang. trunci vagi. This is true for alligators, but in the crocodile some at least of the fibres for the cervical œsophagus pass through the ganglion. If the nerve were cut above the ganglion, stimulation of the peripheral end produced no contraction of the œsophagus, stomach and intestines; but stimulation below the ganglion did, and therefore degeneration extended no further than cells of the ganglion, therefore the fibres ended in the cells and passed on again from them; but never was it possible to influence these fibres reflexly through the ganglion cells. The viscero-motor nerves, to the circular fibres of the hind gut, bladder and uterus, leave the spinal cord in the thoracic nerves and pass down by the abdominal splanchnics to the hypogastric plexus; we do not know to what group of ganglia they lose their medulla. The nerves of the longitudinal fibres of the rectum, bladder and uterus leave by the anterior roots, and pass to the hypogastric plexus in the pelvic splanchnics, where, or later on, they lose their medulla.

The cardio-inhibitory nerves leave the cerebral nervous system in the fine medullated branches of the rami viscerales of the cervico-cranial group; they pass down the vagus to Bidder's ganglion, where they lose their medulla and go on as non-medullated fibres. The vaso-motor cardiac nerves leave by the fine medullated fibres of the thoracic rami viscerales, and go to the proximal ganglia, where they lose their fibres and continue on as non-medullated. Other vaso-inhibitory nerves, as the nervi erigentes and chorda, do not lose their medulla till just before their termination, losing it in the distal ganglia.

The inhibitory nerves of the circular muscles of the alimentary canal leave the cord by the anterior roots, and pass straight to the semilunar ganglia, where they probably lose their medulla. From all the above it appears that the ganglia have three functions. 1. The conversion of medullated into non-medullated fibres. 2. Nutritive effect on efferent fibres. 3. The addition of efferent non-medullated fibres.

The skeletal muscles have the largest medullated fibres—viz., 14·4–18 μ. The unstriped have the smallest, viz., 1·8–3·6 μ; the tongue has medium-sized ones, viz., 7·2–10·8 μ. Therefore Schwalbe's hypothesis, that the size of the fibre is proportional to the length, is untenable. The fact, that involuntary muscles have a double nerve-supply, leads us to expect the same of other structures. In the case of the submaxillary glands this has been proved, the two kinds of gland fibres going the same course as the two kinds of vessel nerves.

In the iris, recent investigations all show there are no dilator fibres of the iris, but it is worked entirely by the circular together with elastic radiating fibres; therefore it is probable that the circular muscle is relaxor as well as contractor. This is strikingly borne out by the fact, that the fibres leave in the thoracic outflow like the inhibitory to the intestine; both pass over the proximal ganglia to the distal (sup. cerv. for the iris), where the medullated fibres lose their medulla.

The cardiac sympathetic nerves belong to the group of vaso-motor nerves, because they augment the activity of the cardiac muscle, and that augmentation is followed by exhaustion. Probably similar nerves exist for all tissues. When tissues are at rest, destruction (katabolism) and construction (anabolism) are equal; hence the nerves, such as motor ones, which augment destruction may be called katabolic. Cardio-inhibitory nerves have a separate existence; they are inhibitory directly up to the muscle; their stimulation is followed by diminished activity, constructive metabolism, repair of function. They may be looked upon as the type of anabolic nerves. One or two experiments are referred to, to show that their stimulation is accompanied by a positive variation.

The author then collects all the evidence which goes to show that the superior cervical ganglia is really one of the distal ganglia, in connection with the central nervous system of the cervical splanchnics. The cells of Clarke's column are exactly confined to those regions of the cord from whence the rami viscerales come. In the case of the spinal accessory there are two roots; the ganglionated fine medullated one comes from Clarke's column, whilst the non-ganglionated comes from the lateral horn and goes to the sterno-mastoid and trapezius; but inasmuch as Clarke's column and the lateral cornu exist elsewhere in the cord, every spinal nerve has three roots: a lateral, which has a ganglionated division connected with Clarke's column, and a non-ganglionated, connected with the lateral column: a posterior which is ganglionated, and an anterior which is non-ganglionated. The anterior and posterior roots, which go to skin and muscles, can be called somatic; the lateral, which is visceral and slightly muscular to skeletal muscles, can be called splanchnic. Then we have a somatic root, with its ganglionated part connected with the posterior cornu, and its non-ganglionated connected with the anterior; and a splanchnic root, with its ganglionated part connected with Clarke's column, and its non-ganglionated with the lateral. It is possible that the rami viscerales may arise partly from the lateral column. It is probable that the ganglia of the sympathetic trunk are homologous to the gang. trunci vagi and form the ganglionated portion of the splanchnic roots. The ganglion of the post. root is probably double, part being connected with the splanchnic and part with the somatic root. The latter part is fixed; the former is not confined to any single position, its component parts are scattered among the various ganglia of the so-called sympathetic system; a small portion still remaining as part of the ganglion of the posterior root. With regard to the question of what muscles are supplied by the non-ganglionated part of the splanchnic root, Bell thought that the respiratory nerves belonged to the same group as the spinal accessory nerve, therefore lateral, like the 11th, 10th, 9th, 7th, and 4th. This is borne out by some observations of Van Wijhe's, that the muscles supplied by this lateral group of cranial nerves are derived from lateral mesoblastic plates, whilst the others come from dorsal plates.

The few large medullated fibres found in the rami viscerales are probably sensory, and as there are two sets of nerves for the skeletal muscles, viz. a somatic from the ant. cornua cells, a splanchnic from the lateral cornu, so there are two sets of sensory, one from the lining of the outside of the body or somatic, one from the lining of the inside or splanchnic; and Schwalbe has pointed out there are two sets of cells in the post. cornu; viz. one in the substantia gelatinosa, and another, the "solitary ganglion cells," nearer the lateral column.

With regard to the cranial nerves. First the anterior roots, 12, 6 and 3, clearly come from the continuation of the ant. cornua cells; 4 seems to belong to the same, from Wan Wijhe's researches; of 7, part clearly arises from same nucleus as 6th. Now the muscles unaffected when the facial nucleus is destroyed are the oc. front and orb. palp. These are clearly somatic, whilst the other 7th muscles are some of Bell's respiratory muscles, and therefore splanchnic, and come from the other part of the 7th nucleus. Certainly most, and probably all the muscles supplied by the above nerves correspond to muscles supplied by the dorsal mesoblastic plates.

Lateral Roots, Cranial Nerves.—All other muscles supplied by cranial nerves correspond to the splanchnic root-muscles derived from the lateral cornu, and are derived from the continuation of it upwards; viz. 5 (motor), 7 (muscles connected with the lateral nucleus), 9, 10 (motor parts), 11 (lat. cornu part), all correspond to Van Wijhe's lateral mesoblastic plates.

The 3rd (lateral root), with its ciliary ganglion; 7th (n. intermedius), with the geniculate ganglion; 9th, with the g. petrosum; 10 and 11, with g. trunci vagi; 12, with g. hypoglossei, are all (except 3rd, which is unknown) in connection with the prolongation upwards of Clarke's column, and, like the splanchnics connected with it, are gangliated.

Lastly, with regard to the post. roots of the cranial nerves, viz. 1, 2, 5 (sensory part with the Gasserian gang.), 8, (with gang. of root), 9 and 10 (with g. jugulare), there are two groups; one ectodermic or somatic, viz. 1, 2, 5, 8, and another endodermic or splanchnic, viz. 9 and 10. As the post. horn ascends, it gets split into two; one part goes to form the nucleus for 9 and 10, and corresponds to the solitary ganglion cell-group, which therefore might be called splanchnic; and the other part forms the nucleus of 8 and 5, and is a continuation up of the substantia gelatinosa, which might therefore be called somatic. The author concludes with a table which shows all the various cranial nerves in their relation to the medulla oblongata, hind-brain and mid-brain respectively.

W. Hale White.

Abstracts of British and Foreign Journals.

On Paralysis during and after Pregnancy—*Ueber Paraplegie in der Schwangerschaft.* By Professor Jolly (*Archiv für Psychiatrie*, Bd. xvi. Heft 3).—A girl, æt. 16 years, became completely paraplegic by the third month of pregnancy. This condition continued till the birth of the child, which took place naturally, and at full time. Fourteen days later, on her coming to the hospital, she presented the following condition: paralysis, both lowers, with well-marked knee-reflex and considerable diminution of sensibility, especially on the left side; lowers cold and cyanotic. After six months' treatment with the faradic current, the patient got better, but trembled all over, like an insular sclerotic. Since then she has had aphonia, with paralysis of a vocal cord. Ultimately all the symptoms disappeared and the patient completely recovered. The author discusses the possibility of its being a reflex paraplegia, due to pressure on the sciatic nerves, or to actual change in the cord; he comes to the conclusion, that it is a functional disorder.

Puerperal Endocarditis, with Hemiplegia after Delivery. By W. C. Neville, M.D. A paper read before the Obstetrical Section of the Academy of Medicine in Ireland.—E. C., æt. 30, seventh confinement, labour normal. The day after confinement, whilst sitting up in bed, suddenly felt giddy and fell back "convulsed," and on examination was found to be completely hemiplegic on left side. Two days later, a to-and-fro basic murmur was detected. Four days after this, the secretion of milk stopped, the temperature began to rise, and septic symptoms showed themselves; and a fortnight after delivery the patient died. At the autopsy there were discovered recent vegetations on the aortic valves, with embolism of the right internal carotid at the base of the brain, with softening of much of the brain supplied by that vessel.

The writer points out that although many theories have been put forward to account for the paralyses of pregnancy, it has lately been shown that, in those cases at least which die, the paralysis is due to embolism.

Des Manifestations Méningitiques et Cérébrales des Oreillons, par les Drs. M. Lannois et G. Lemoine (*Archives de Neurologie*, Jan. 1885, vol. xi. No. 31).—This paper contains a very good bibliographical account of the subject of meningitis in the course of mumps. In the first part of it the authors refer to many published cases, in all of which, however, the symptoms of cerebral disturbance, such as delirum and coma, did not last long; but the chief part of the communication is the second section, in which accounts of cases are given in which an effect lasting over many months remained.

After quoting cases of paralysis and aphasia, following mumps, and extending over periods of six months and fifteen months, they record their own case, in which, three days after recovery from the mumps, the patient, a man in a regiment, was suddenly attacked with vomiting and coma. He understood when he was spoken to, but answered unintelligibly, and quickly relapsed into his drowsy state. He also had right hemiplegia, with right complete anæsthesia, and some contraction in the extended position. The left side of the face was paralysed; there was left conjugate deviation, and the tongue was inclined a little to the left; he had headache and dilated pupils; for the first few days of this attack there was fever. He gradually got better, the face and eyes recovering first, and later on the aphasia and paralysis; but there remained some difficulty on these two points for a very long while, but when seen a year after, he was quite well.

In most of the cases orchitis was present, and as a rule these symptoms come on after the subsidence of the orchitis and parotitis.

The authors then discuss the possible causes of this complication of mumps. They dispose of the suggestion of metastasis as meaningless; of that of pressure in the neck, for the swollen parotid is often on the wrong side; of embolism, as being unable to explain the cortical symptoms; of thrombosis, as not explaining the multiplicity of symptoms. They conclude that it is probably a meningo-encephalitis, the former being primary, and that the hemiplegia and aphasia are due to a large amount of exudation at the necessary places. It is to be regretted that the authors did not wait before the publication of their cases for the death of the patients, for, as the matter stands, their hypothesis is absolutely unsupported by any evidence, and it is extremely improbable that all their cases, if due to meningitis, should recover, as this result is very rare; or that they should all have aphasia and hemiplegia present for so long a time, as these are both rare sequences of the usual forms of meningitis. None of their cases were in children.

HALE WHITE, M.D.

On Paralysis of Old People, and their probable relation with Uræmia.—Dr. RAYMOND (*Revue de Médecine, No.* 9, 1885), remarks that till recently it was a sort of clinical axiom that uræmic cerebral phenomena were necessarily bilateral, and that being produced by a toxic influence, they could not manifest themselves by any localised symptoms. In this article, Mr. Raymond records eleven cases (in ten of which autopsies were made), tending to prove that uræmia may give rise to localised symptoms.

He groups these cases into three classes: in the first, an individual apparently in good health (and almost always of advanced age) becomes suddenly apoplectic, and dies forthwith; or, after emerging hemiplegic from the coma, dies a little later.

In a second class of cases, sudden coma occurs: the patient recovers from this, but is left with a hemiplegia—in some cases accompanied by rigidity and contraction—and hemianæsthesia; he remains in this condition until he is again seized, after a variable duration of time, by well-marked uræmic symptoms, which quickly prove fatal.

In M. Raymond's third class of cases, a patient who has suffered at some previous date from some focal lesion, the symptoms of which have entirely passed off, becomes the subject of uræmia. He then manifests convulsive movements entirely limited to the side which had been previously paralysed. The autopsy in such cases shows no *recent* change capable of explaining the unilateral motor symptoms.

In all these cases a fibrotic and usually a contracted kidney was found post-mortem, with frequently evidences of an additional more recent subacute nephritis: in some cases there was hypertrophy of heart, and usually atheroma or thickening of the arteries in the cranial cavity.

In all there was well-marked œdema of the brain, with serous effusion into the ventricles and into the subarachnoid space, a condition formerly described as serous apoplexy.

According to M. Raymond, the serous effusion is the consequence of uræmic poisoning, itself the result of the Bright's disease. The effusion compresses the cerebral arterioles, and as these vessels in aged people are already weakened by degenerative changes, a very slight pressure upon them suffices to disturb the regularity of the circulation in the brain-substance, and to produce paralytic or other effects. Further, he suggests that in some cases arterial degeneration affects some vessels more than others, and that those the least weakened by disease are able to resist the pressure of the surrounding serous effusion, while those more extensively degenerated yield to its pressure.

In consequence "ischæmia" will be produced in corresponding areas of brain-substance, and will be evidenced clinically by hemiplegia or unilateral convulsions. In the case of the subjects of an old cerebral hæmorrhage or softening, although the resulting *symptoms* may have passed off, the area of brain around the lesion remains in a condition of relative bloodlessness, and then the slightest disturbance of intracranial pressure suffices to still further increase this, and paralytic or convulsive phenomena are produced similar to those shown by the original lesion.

M. Raymond gives experimental proofs of his theory. After removing the inferior cervical ganglion on one side of a rabbit, so as to destroy the vaso-motor nerve supply of one of the cerebral hemispheres, he tied both ureters, so as to produce a uræmic condition. Convulsions ensued, limited to the half of the body opposite to that on which the cervical ganglion had been removed. In another set of experiments practised on the dog and on the rabbit, he destroyed a portion of the cerebral cortex, producing paralysis in *one* limb, which gradually passed away. He then tied the jugular vein on the same side (an operation which in the dog usually produces an incomplete and transient hemiplegia) and a well-marked hemiplegia resulted: the fore-limb soon recovered its power; but the hind-limb, which had been the one to suffer from the previous removal of brain-substance, remained permanently paralysed.

Post-mortem, only a general œdema of brain was seen, especially pronounced in the region around the removed brain-matter.

M. Raymond's observations and experiments lead him to the following conclusions:—

1. Fatal attacks of apoplexy occur in aged persons, or attacks followed by hemiplegia of similar character to those from hæmorrhage or softening.
2. These attacks are due to the uræmic state.

3. Post-mortem, a general œdema of brain is found, or an old focal lesion.

4. The apoplectic attacks and the hemiplegia are the results of the œdema of the brain, and the disturbance in cerebral circulation produced by it. These circulatory troubles resolve themselves into want of oxygen in the cerebral blood.

5. A patient suffering from chronic renal disease, and who has suffered from a former hemiplegia, may, when he becomes uræmic, suffer from convulsions limited to the side of the former hemiplegia.

A Case of Paralysis Agitans produced by Damp and Cold.—Dr. Boucher (*Le Progrès Médical*, 25 Juillet 1885) describes the case of a female, married, aged 50, in whom the characteristic tremors of paralysis agitans commenced suddenly on the right side of the body; soon after, the left side became affected; the usual symptoms rapidly succeeded, and in eighteen months from the commencement of the tremors, the rigidity of the trunk and limbs, with flexion of the neck and of the various joints, became so great that the patient would not show herself in the street, for fear of ridicule. There was diminution of muscular power, as shown by the dynamometer, and much suffering from an almost insupportable sensation of heat. She could, however, sit close to a fire and bear the heat from it, when others could not approach it. Her general health was fairly good.

M. Bouchard remarks on the more frequent occurrence of paralysis agitans in England and France than elsewhere in Europe, and refers to the experience of M. Charcot that the disease especially affects the Anglo-Saxon race.

He ascribes the disease in the patient to her having for a long period previous to its onset been living in a damp house, and to her having been accustomed to devote several hours a week, throughout the year, to washing linen in an outhouse much exposed to currents of air.

The patient was treated by bromide and by iodide of potassium; improvement occurred during the administration of the latter, but he does not ascribe this to the use of the drug.

On Uræmic Hemiplegia and Partial Epilepsy.—MM. Chantemesse and Tenneson (*Revue de Médecine*, No. 9, 1885) combat the idea that uræmic poisoning can never show itself by localised symptoms; they point out that other toxic agents, such as lead and mercury, may attack the more vulnerable parts, and produce localised motor or sensory effects.

They record notes of six cases, in all of which there was either a local paralysis or Jacksonian epilepsy. In five an autopsy was obtained, but nothing was discovered except long-standing renal disease and general serous infiltration of pia mater and brain-substance. The hemiplegia was in two cases right-sided, in two cases attacked the left side; the face and limbs were affected; the paralysis varied in amount from time to time, but gradually tended to pass away partially or completely.

The loss of sensibility was always less complete, and hemi-anæsthesia was only appreciable when loss of motor power was very well marked.

Twice conjugate deviation of eyes and rotation of the head were present in two cases, once towards, once away from the paralysed side.

In two cases one-sided epileptic attacks occurred, coming on without warning, and affecting face, arms and leg, but unaccompanied by any intellectual disturbance. The attacks lasted a few minutes; they recurred several times daily for a week, till death occurred. In one case—that of a woman who became albuminuric during pregnancy — the hemiplegia was preceded by violent neuralgia of the 5th nerve and the brachial plexus.

The "albuminuric hemiplegia" may persist several days with little observable change till death, or may pass off gradually to disappear altogether, or sometimes to recur. What is most characteristic is a tendency to vary in amount—thus the hemiplegia may pass off in the morning and return a few hours later in the day.

During the paralysis the ordinary signs of uræmia are absent, except the urinary changes. There is no headache, no vomiting or digestive disturbance, no anasarca, no lung affection. But the peculiar whiteness of skin of albuminurics and contraction of the pupils will render the diagnosis of uræmia almost certain. In no case was the fall in temperature observed which usually accompanies uræmic coma; on the contrary, on the onset of the symptoms the temperature always rose, and towards the fatal termination ran up as high as 40°–41° C.

Post-mortem examinations in the five cases only revealed a diffuse chronic interstitial nephritis, congestion of cerebral vessels and œdema of brain-substance.

The onset of the symptoms by an apoplectic attack, the localisation of the paralyses to one side of the body, with slight anæsthesia, and the rise of temperature, might all lead to a suspicion of softening from occlusion of vessels; but the myoses, the low sp. gr. of the urine and the albuminuria, will suggest uræmia.

As the two conditions do not exclude one another, the diagnoses in old persons may be impossible. In young patients the myoses, the visual disorders, and the abundant albuminuria which precede the paralyses, the variations in degree of the latter, with possibly a little weakening or stiffness of limbs on the opposite side of the body, will make the diagnosis of uræmic poisoning.

From actual cerebral hæmorrhage the diagnosis is easier, as there is an immediate fall in temperature, and the paralysis is more permanent.

In one of the cases recorded, one-side epileptic attacks occurred several times daily in a man of 35, who had had syphilis: post-mortem, only serous effusion was discoverable; in such a case cerebral syphilis might have been diagnosed, but for the albuminuria and the facts pointed out.

MM. Chantemesse and Tenneson point out, that these cases present different symptoms to those usually produced by uræmic or other toxæmia: the temperature is raised, the paralysis is localised, and coma may be absent; and indeed the symptoms are those not directly of toxæmia, but of a mechanically acting lesion, either structural or functional. They do not agree with M. Raymond that atheroma of arteries must be a necessary precedent for local œdema from uræmia, for three of their patients were under 35 years of age, and the autopsies showed the absence of degenerative changes in the cranial vessels.

They suggest that these local affections are analogous to the sudden œdema of eyelids, and to the local transient patches of œdema or congestion which occur in the lungs of patients with Bright's disease.

In conclusion they affirm that, contrary to received opinion, a hemiplegia without rigidity and of sudden onset, just as is producible by hæmorrhage, may result from uræmic poisoning, and in some cases unilateral epileptic attacks are also produced. These phenomena must be connected with a circumscribed œdema, or at any rate by an œdema which predominates in one part of the brain, and such œdema is the consequence of a blood-alteration, and is of uræmic origin. SIDNEY PHILLIPS, M.D.

Two Cases of Acute Traumatic Myelitis. By Dr. H. D. SCHMIDT. (*Journ. Ment. Dis.*, New York.)—(Abstract.)—These two cases have a somewhat similar history, both patients having received an injury affecting the cervical region, and followed by the symptoms of acute myelitis; death in one case occurring six days after the injury, in the other the following day. At the autopsies—in each case the spinal cord was found constricted in the cervical region, this constricted portion being bounded above and below by a hernial-like swelling (caused by displacement of tracts of nerve-fibres, chiefly of the white lateral columns). On microscopic examination (of teased-out fragments of the constricted portion) the nerve-fibres were found distorted and injured. There were a number of ganglionic bodies with or without processes or their fragments, either disintegrating or having undergone fatty degeneration. Among the nervous elements were numerous capillary hæmorrhagic centres, fragments of small blood-vessels, or blood-corpuscles disseminated throughout the tissues. The question here arises, How far were these appearances due to the injury, or how far due to the method of preparation?

On examination of those parts of the cord bordering the constriction, all the vessels of the pia mater were found congested, and not only filled with blood-corpuscles, but in the small cells in the walls of these vessels there was an increase of the protoplasm. The minute vessels of the cord were in the same condition, and here the increase of the protoplasm and its subsequent fatty degeneration with that of its nuclei had, in some instances, assumed enormous proportions. The ganglionic bodies appeared to be in a state of parenchymatous inflammation, and in a few there were indications of commencing degeneration. The most striking changes, however, were observed in the nerve-fibres of the white columns, a great number of the axis-cylinders being more or less swollen, some even to an enormous extent, and still surrounded by their medullary sheaths, or laid entirely or partially bare.

Although these swollen axis-cylinders still showed their granular anatomical composition, the regular linear arrangement of the minute granules was lost. The medullary sheaths of almost all the nerve-fibres in these preparations were deranged, their fibrillar layers being displaced to form larger or smaller varicosities, whilst their fibrillæ appeared separated.

The difference between the two cases was one of degree or extent of the inflammatory process, and manifested itself in the condition and dimensions of the swollen axis-cylinders. Thus, whilst the swellings of the axis-cylinders of the nerve fibres in the cord of the second case had, in the short space of fifteen or sixteen hours, only advanced to form a simple varicosity; in the first case, lasting more than six days, the same swellings had extended more or less in a longitudinal direction over the axis-cylinders, and fatty degeneration had made its appearance.

These descriptions are accompanied by fifteen illustrations.

On Morbid Drowsiness and Somnolence. By C. L. Dana (*Jour. Neur. Ment. Dis.*, New York, 1884).—Dr. Dana classified the forms of morbid somnolence as follows:—

1. Epileptoid sleeping states.
2. Hysteroid sleeping states, including
    (*a*) spontaneous "mesmeric" sleep.
    (*β*) trance, and lethargic states.
3. Morbid somnolence, the expression of a distinct neurosis (narcolepsy).

Illustrating these different forms were the reports of fifty cases, of which eleven were of Class I.; fourteen were of the "hysteroid" and twelve of the "mesmeric" form of Class II.; whilst nine of the remaining cases were classed under morbid somnolent neuroses; the few additional cases being considered apart, as arising from organic brain-disease, exhaustion, diabetes, &c.

The somnolence showed itself in a variety of ways; either, (*a*) by prolongation of natural sleep, the attacks daily succeeding each other; (*β*) by constant persistent drowsiness; (*γ*) by comparatively brief attacks of somnolence with no drowsiness in the intermissions; (*δ*) by single or repeated lethargic attacks (as in the cases of the famous sleeping and fasting girls); or, lastly, (*ε*) by periodical attacks of profound somnolence or lethargy.

Five cases came under Dr. Dana's own observation. The first was that of a young man of healthy family personal history, who gradually developed the habit of prolonged somnolence. The second was that of a young lady who was subject to short attacks of catalepsy, cataleptic petit-mal, alternating with sudden attacks of sleep, these attacks coming on several times daily. The other three cases were of neurasthenic persons, who, for several months, had persistent drowsiness not attributable to any nutritive or organic disorder.

Briefly stated, the causes assigned were: (1) old age, when there is a weakened heart or diseased arteries, with cerebral malnutrition; (2) the diseased vascular conditions preceding cerebral hæmorrhage; (3) the cerebral mal-nutrition or inflammations preceding or occurring during certain forms of insanity; (4) various toxæmiæ, *e.g.* malarial, uræmic, cholæuric, syphilitic; (5) dyspepsia; (6) diabetes; (7) obesity; (8) insolation; (9) cerebral anæmia and hyperæmia; (10) cerebral tumours and cranial injuries; (11) exhausting diseases; (12) the sleeping sickness of Africa.

At subsequent meetings of the New York Neurological Society, additional cases of somnolence were reported as occurring in locomotor ataxia, chronic endarteritis, myxœdema, diabetes, syphilis, epilepsy, &c. Dr. Morton related an interesting case in which the patient, a physician, suffered for fifteen years from an inordinate sleepiness, which could not be overcome by mental effort, this mental lethargy lasting for many hours at a time.

T. Bulkeley Hyslop, M.B.

Dr. Alex. Thom on a Case of Left Facial Paralysis (*Edin. Med. Journal*, July 1885).—A description is given of the case of a boy, aged 9 years, with a neurotic and strumous family history, who came under the author's care complaining of vomiting, occasional headache, staggering gait—not due to giddiness—and inability to retain the head erect, the symptoms being of three weeks' duration. He presented double optic neuritis, paralysis of the left side of the face and palate, left internal strabismus and nystagmus (apparently unilateral), paresis of the left arm and leg, with diminished knee-jerk; there was impairment of the sense of hearing on the same side, and some difficulty in swallowing; the urine contained a trace of sugar. The faradic excitability of the paralysed muscles was markedly diminished, and their galvanic excitability augmented without qualitative alteration. Sloughing of the left cornea, with loss of sensation over that side of the face, supervened; speech became impaired; paralysis extended to the right lower extremity, and finally to the muscles of respiration, causing death about ten weeks after the development of symptoms. After careful discussion the author concludes that the lesion was tubercular in nature, originated in the medulla at the seat of the nuclei of the sixth and seventh nerves, and by its extension implicated the pyramidal fibres of the opposite side, and the middle peduncles of the cerebellum. Unfortunately, the absence of a post-mortem examination deprived the case of its value. A statement, that the electrical examination showed that the lesion was not situated in the nerve-trunks, seems hardly justified by the author's description.

Westphal on a remarkable Case of Periodical Paralysis of all four Extremities, with Abolition of Electrical Excitability during the Paralysis (*Berliner Klin. Wochenschrift*, 1885, Nos. 31, 32).—A well-nourished and healthy-looking boy was admitted into Professor Frerich's Clinic on January 7th, 1885. His parents were healthy in every respect, and no member of his family had suffered from nervous disease. Four years previously he had had severe scarlatina, followed by another illness, probably Bright's disease. Four weeks after complete recovery he complained for one day of "curious sensations" in all the limbs, tingling in the hands and pains in the feet, and in the course of the following night he suddenly lost all power of movement; he perspired freely, and experienced great thirst and heat of skin. Similar attacks had recurred, at first at intervals of 4–6 weeks, subsequently with increasing frequency and severity, till the date of his admission. Previous to his transference to the Nerve-Clinic, on January 30th, he had three paralytic attacks, in which the faradic excitability of the muscles was found to be abolished; but the observation was considered to be of doubtful accuracy.

He had fresh attacks of complete paralysis of the extremities, but of only a few hours' duration, during the nights of February 4th and 10th. Dr. Oppenheim then undertook the investigation of the case, which was rendered difficult by the invariable occurrence of the attacks at night. On March 16th the patient began to complain of weakness in the right knee at 4 P.M.; at 5.30 P.M. he fell to the ground, and complained of pricking pains in both legs and feet, of great thirst, and of desire to micturate, but could only do so after an interval of some minutes. At 7 P.M. there was still a little power of voluntary movement at both hip-joints, and at the left knee and ankle; all the movements of the arms were weak. At 4 P.M., on March 17th, Dr. Oppenheim found him lying on his back, his mental faculties intact, complaining of some difficulty in rotating his head, and coughing and sneezing. Paralysis of both upper and lower extremities was complete, without rigidity or contracture; sensation everywhere unimpaired, knee-jerks present, plantar reflex abolished, but cremasteric and abdominal reflexes natural. On electrical examination, only very faint muscular contractions were obtained with the strongest faradic current over the nerve-trunks of both arms and legs, the patient experiencing great pain from the examination; the excitability of the left peroneal nerve and of the muscles supplied by it was completely abolished. At 8 A.M. voluntary power had to some degrees returned in the muscles of the arms, which reacted comparatively well to direct and indirect faradic and galvanic stimulation; the legs were still paralysed, but the muscles of the calf reacted normally to direct galvanism, and the peronei very slightly to strong faradism. At 9 A.M. the power of flexion and extension of the fingers had returned, and some slight power of extension of the feet and toes; the head could not be flexed towards the sternum, the knee-jerk on both sides was very feeble. At 1 P.M. the patient had regained all voluntary movements, but those of the legs were still very feeble, and the faradic excitability of the extensors of the feet greatly diminished. Throughout the attack there was no impairment of sensibility, no affection of the muscles of the face or eyeballs, no loss of muscular sense, no enlargement of spleen. His evening temperature in the rectum was 39° C.; the urine contained nothing abnormal. On the following day he perspired freely, and complained of heat of skin; the faradic excitability of the extensor muscles of the left foot was still very markedly subnormal. On April 9th at 2 A.M. he had another attack, similar in all essentials to that described, but with total loss of knee-jerks and of shorter duration, both voluntary movement and faradic excitability of the muscles being re-established in seven hours. The patient had a third, milder attack, on April 16th, and was removed by his parents from hospital on April 23rd. He continued to have frequent similar attacks till the end of June, when he was lost sight of.

Westphal indicates in regard to his case the points of divergence from, as well as resemblance to, others described as "intermittent paralysis," by Romberg, Cavaré and Hartwig, and attributed by them to malarial influence. In Romberg's case[1] the attacks were quotidian in type, and yielded to quinine; ague was prevalent, but the patient had not suffered from it. Cavaré's case,[2] also quotidian in type, occurred in a nervous young woman three days after parturition, and was arrested by quinine; but there is no statement as to the co-existence of malarial fever. Hartwig's case[1] is narrated in detail. The patient was a robust man, aged 23, who had had tertian ague five years previously. In the autumn of 1873 he began to suffer from intermittent, total paralysis of the muscles of legs, trunk, arms, and neck, with impairment of speech, deglutition and respiration, beginning at night, unaccompanied by fever or sensory disturbance, but attended with copious sweating, and recurring every twenty-four hours. In hospital the condition became aggravated, the attacks lasting 40 hours, the intermissions only 6–8 hours; full doses of quinine apparently warded off the attacks. In March 1874 he had a fresh attack, during which the reflexes were abolished, and the excitability of the paralysed muscles almost completely so. Although quinine at first seemed to prevent the attacks, these soon returned, and, presenting usually a tertian type, persisted till his discharge about eight months afterwards. In the intervals considerable paresis of the muscles, with contracture and diminished electrical excitability, was present; on only one occasion was there a rigor and rise of temperature to 40° C. As in Westphal's case, the attacks were apparently sometimes warded off by movement, and the paralysis, which commenced in the lower extremities at the periphery, extended to the trunk, afterwards in the upper extremities, spreading from the trunk to the periphery. Recovery in both cases began at the periphery, first in the arms, afterwards in the legs.

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[1] 'Lehrbuch der Nervenkrankheiten,' 3 Auft. 1857, p. 752.

[2] 'Gaz. des Hôpitaux,' 1853, No. 89. Same case is referred to by Erb ('Handbk. d. Krankh. des Nervensystems,' i. p. 322) as Macario's case.

Westphal, whose own case presented nothing of a periodic or intermittent character, doubts whether any of these cases can be justly attributed to malaria, and does not consider their improvement under quinine as conclusive evidence. At all events, the electrical conditions in his case and in Hartwig's are quite inexplicable. No known disease of spinal cord or of spinal nerves has been observed to cause similar phenomena; nor can a sudden nutritional disturbance of muscle or of nerve-endings, due to circulatory derangement, be invoked, as the state and temperature of the skin, and the perfect integrity of the sensibility, lend no support to such a view.

J. J. Pringle, M.D.

BRAIN.

OCTOBER, 1886.

Original Articles.

ON THE EXCITABLE AREA OF THE CORTEX, AND ITS RELATIONS TO THE COLUMNS OF THE SPINAL CORD.

A Reply to Professor HORSLEY.

BY MAURICE SCHIFF, M.D.,

Professor of Physiology in the University of Geneva.

FIFTEEN years ago, when repeating the experiments of Hitzig and Fritsch on the excitable area of the cerebral cortex, I found some new properties of this cortex, and gave a new explanation of the well-known results of its electric excitation. These facts, previously announced in the[1] 'Giornale Imparziale Medico' of Florence, were described first in the appendix to the second edition of my 'Lezioni di Fisiologia Sperimentale,' Florence, 1873, p. 523–540, and later in the first part of the 'Rivista Sperimentale di Frenatria,' for 1876, and in Pflüger's 'Archiv f. Physiologie,' vol. xxx. (1883) pp. 212–275, and in the same journal, vol. xxxiii. (1884) pp. 264–271.

I cannot repeat all, nor even the principal part, of what I said in the above quoted papers; but it seems to me that Prof. Horsley, before undertaking the criticism of my views on the subject, should have made himself thoroughly acquainted with these memoirs, where, I believe, I have plainly justified my opinions. He should not have directed his criticism against a single paper by Dr. Huggard, that was not intended to demonstrate the reality of my theories, but merely to record some facts, which had struck the author whilst working in my laboratory.

I encouraged Dr. Huggard to publish his paper, not only because it contains some facts which could not yet be known to English readers, but also because I hoped that it would draw attention to my former publications, where the views to which he alluded have been justified by experiments, and in which my experimental methods are sufficiently set forth. If Prof. Horsley, after having read and repeated my experiments, still retains some doubt as to the reality of the facts, or to the validity of the conclusions, I am ready to enter upon a discussion with him, and to correct the errors that he can prove I committed, either in my method or in my reasoning.

But for the present I can only restate the most general results of my researches, giving merely one or two examples of the facts that have determined my judgment.

Symptoms of the complete destruction of the Excitable Area.

All the experiments I shall mention were made on the dog, when no other animal is specially indicated. The conclusions therefrom refer to the dog. In researches on the central nervous system, I never venture to generalise results obtained on one or two species of animals only. For other animals, such results point to possibilities which we must keep in view, but cannot take as realities, unless confirmed by special experiments. Hence I trust the reader will be able to accept the results, without being prejudiced by clinical or experimental facts, resulting from observations made on higher animals.

When we endeavour in a series of dogs to effect the destruction of all the excitable cortical area, we never can make in every case the conditions so exactly alike, that absolutely the same result is obtained in the early days after the operation. At this period traumatic irritation still exists; but after we have reached the stage which I have called "traumatic equilibrium," we distinguish certain symptoms which are uniformly present *in all the cases*; whilst others are shown only by animals with a deeper, more extended destruction of the cortical and a part of the white substance.

So we must distinguish traumatic from permanent symptoms; and among the permanent, those which are essential and general from those which, accompanying the essential ones, are present only in a certain number of cases.

I shall not enter here into an enumeration of the traumatic symptoms, observed before the so-called "equilibrium" has been reached; it may be sufficient to say, that in the first period the essential symptoms are not wanting, but they are complicated by others, which disappear after a certain time, varying from some hours to three weeks. It is evident that, contrarily to the views expressed by some authors, these do not depend upon the destroyed organs, but upon others, which are *probably* in the proximity of the wound. I take it for granted that the function of a destroyed organ is lost for ever, and cannot be re-established by compensation. It is not the loss of a function, but only the secondary effect of that loss, that can be diminished by the exercise of other organs. Hence we must expect that the true function of the cortex is lost for ever after its destruction, and that all such symptoms as disappear, either wholly or in part, are due *not to the loss of the organ*, but to the effect of traumatism. I must insist on this principle, which has been very often forgotten in the discussions on the functions of the brain.

Among the symptoms that disappear in the early period, sometimes within the first hour, after the operation, after the dog has recovered from the ether narcosis, is a diminution of all the sensations in the limbs, produced by pricking with a needle, by pressure, or by traction of the muscles. These sensibilities are not entirely lost (as sensibility for touch and cold really is), but they are feebler than in the normal state, feebler than on the corresponding parts of the other side of the body, even when on this other side there is no hyperæsthesia.

This state of the sensation (tactile anæsthesia) produces, on the side opposite to the operation, a surprising indifference to the positions of the limbs. This indifference has sometimes been taken for an expression of paralysis or paresis. It is much more evident when the dog is at rest, there being no motor current; here it cannot be *produced* by the weakness of this motor nerve-current. When the dog is walking, he shows in a certain degree, the appearance which I have described on page 143 of my 'Physiology of the Nervous System' (Jahr. 1858–59), as the effect of the section of the posterior spinal roots, with perfect conservation of motor energy. And these observations, made first at Berne in 1856 and 1857, have been repeated and confirmed within the last few months by Dario Baldi[1] in a very interesting series of experiments, in which he was able to preserve for a very long time the dogs that had been operated upon. It is singular that this author, who arrives at no new conclusions, does not mention the experiments of Panizza, nor of Stilling, nor of myself, which have since been repeatedly demonstrated in the laboratory of Florence, where Baldi himself is now working.

When we stretch a limb, or bring it in a very inconvenient position, the animal—unless the equilibrium of its body be disturbed—does not resume its attitude before walking or making a general movement. Can this dog make a voluntary movement with a single limb, unless under the influence of the performance of a combined movement due to a spinal mechanism, like that of walking?

If I can prove that an intentional isolated movement is still possible, the want of such a movement in the case we consider here, can only be attributed to the want of intention. And the absence of intention can only be due to the fact, that the tactile insensibility, the existence of which is evident from other experiments, hinders the dog from being aware of the irregular position of his limbs.

A dog in which the state I am now describing lasted for six hours, was lying on the floor with the anterior left extremity stretched out at right angles with the body. I brought a metallic electrode, connected with a small Ruhmkorff coil, near the ball of the foot, and discharged three times into it a unipolar spark. After every discharge the foot was removed and brought forward, and hidden under the neck of the dog. I replaced the limb in its former stretched position, where it remained quiet till the following discharge. At the fourth time, the animal, seeing the movement of my hand and the electrode, did not wait till I was close to the foot, but before the discharge occurred, removed the limb to hide it under his neck. Thus it is clear that a voluntary movement, a movement due to a mental impression, and not to the action of a general mechanism, was possible. It is exceptional that, as in this case, a cerebral movement occurs so early; but this exception is not a very rare one. I must remark, that the local impression which produced here a movement by a simple recollection was not of an extraordinary strength, because the dog was not put to flight, nor to a general movement; while the latter effect could easily be produced by firmly pinching the tail with my fingers. The phenomenon persisted for some weeks, till the same dog could almost give the paw when ordered.

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[1] Baldi, 'Lo Sperimentale;' Florence, 1885.

The indifference to position is but the effect of insensibility.—When at a later period this insensibility is more and more reduced to want of sensibility for touch and for cold only, the indifference is less marked, though it persists indefinitely for those abnormal positions which the normal dog perceives only through the sensation of touch. And in the same manner the want of movement is reduced to those movements, which are normally excited by touch or by cold.

When the traumatic equilibrium sets in, the former are very quick, the latter become slower and slower. But before and during the return not a single muscle is paralysed, nor a single movement impossible. This is a point in which even Goltz and Munk, who were not always of this opinion, have for some years agreed perfectly with my observations. Lautenbach, who had assisted at some of my experiments, has published a paper in order to defend the same opinion. I must insist on the fact, that most of the dogs in which all the excitable cortex has been completely removed, begin after some weeks to use their anterior extremities as a hand. They give the paw at our request, if they have formerly been taught the trick. They hold a bone or a hard piece of bread, or rather they try again to hold it with the two anterior extremities. Cats, squirrels, and rats climb with the aid of the two or four extremities, and they place them, not as if they were directed by a pre-established mechanism, but in accordance to information given by visual and by central impressions generally. I have already insisted on all these points in my first-quoted paper in Pflüger's 'Archiv,' where I have explained the remaining imperfections of these intentional movements by the loss of the direction given by the different organs of tactile sensibility. By reading this paper, Prof. Horsley may easily be convinced that the hiatuses he speaks of ('BRAIN,' *l. c.*, p. 45) exist less in my description than in what he knows of my observations. Most of the characteristic features with which Prof. Horsley tries to fill up these hiatuses must be again put out of the way, if we wish to know the real facts, and not Horsley's ideas about the facts.

It is true that in some, but far from all, cases, certain intentional and isolated movements of the fore and even of the posterior limb, did not return after several months. These are the cases in which I think (see Pflüger's 'Archiv,' 1883) that not only the cortex, which is only a channel for tactile (and thermic) impressions, but that the *centre* for these sensations, has also been damaged. I assume here a greater extension of the extirpation, though I must say that post-mortem examinations have not yet justified this opinion. I have found cases of this second category, in which the wound examined with the naked eye was only some millimetres deep, and have seen two examples of the first category, in which intentional movement returned, and in which the wound extended deeply into the brain, so as to destroy the upper part of the lateral ventricle. These examples prove, that the *loss of intentional movements is not one of the essential effects* of the complete destruction of the excitable area of the cortex.

Though the dog uses his anterior extremities for holding the food, he does not use them well. At first he makes the suitable movements to touch or to fix the bone. But while he is eating he must be guided by touch, in order to hold fast when he makes traction with the teeth. Our dog cannot feel the traction ; he cannot feel whether he touches or not, and after a short time, still keeping his fore-paws fixed, he touches no more. The flexion of the toes, not being limited by the resistance of the object, becomes exaggerated, the paw is bent and kept in flexion. After a while we see the paw held in the same position, but at a certain distance from the object, the dog not being aware that he is no longer touching it. Strength is not wanting, nay, at the beginning of the movement is sometimes *stronger* with the insensible paw; as can be shown in dogs, and better still in squirrels and cats, by offering them a piece of dry or half-dry bread hanging on a string. The impressions of the toes and claws of the insensible side, which cannot hold fast, are often more visible and deeper than the impressions of the other side.

As I said as early as 1858, in my 'Physiology of the Nervous System,' the movements which are not guided by sensibility are as often too strong as they are too weak. The same rule holds good for the strength of the movements with which the toes are placed on the ground.

It is more than ten years since I investigated this point by moistening the under side of the foot with oil, and observing the prints left by the animal walking along my long corridor in Florence. The sound produced by the contact may, on a resounding ground, give the same evidence in dogs, and still better in squirrels.[1]

When a dog or a cat is shaking itself (for instance, after having been sprinkled with water), the insensible extremities cannot quickly or adequately moderate and regulate the contact with the ground; they slip, and the animal may fall on the same side. On this point we must correct what Prof. Horsley (*l. c.*, p. 45) says of the position of our dogs that have been operated upon.

Prof. Horsley believes he must draw my attention to the position of the toes. But the possibility of making the dog or the cat stand with the toes of the anæsthetic side doubled up, so that they really stand, and somehow walk, on the dorsum of the foot, has been for me for a long time[2] the simplest and most evident sign of anæsthesia of touch. For many years I used to make my students try for themselves to double up the toes of the two sides, and feel the difference in the resistance. But I must add to what has been said by Prof. Horsley, that, when the dog has returned to perfect traumatic equilibrium, he no longer, or very seldom, takes this position by himself; like an ataxic man, he accustoms himself to a stronger tension of the muscles, the movements of which he no longer feels.[1] But we can produce the aforesaid position with our hands, when the dog is standing quietly on the table, and observe the resistance of the dog, and the persistence of the position. I believe I have clearly and sufficiently proved[2] that the non-resistance of the dog to this tactile excitation does not depend, as Prof. Horsley still believes, on a paralytic state, and that there exists no trace of paralysis, either in the extremities or in other muscles of the body.

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[1] See Pflüger's 'Archiv,' 1883, p. 435.

[2] See my 'Lezioni di Fisiologia,' 2nd edition; Florence, 1873, p. 537.

We can prove in an analogous manner, as I did it in the last quoted paper for the toes, the non-existence of paralysis and the existence of tactile anæsthesia in one half of the head, opposite to the decortication of the brain. This has been shortly indicated, as early as 1873, in my 'Lezioni di Fisiologia,' p. 538.

Should I repeat here all that I have published on that matter, this paper would assume the length of a large volume; and such a repetition would be useless. I believe I have said enough to convince Prof. Horsley, that he must study the original articles and those of Goltz and Munk, if he wishes to have an idea of the actual state of our knowledge on this subject. As I said in the beginning, Dr. Huggard's valuable articles (and Prof. Horsley seems to know only these communications, which are and must be fragmentary) contain only the indication of some new facts, which must be misunderstood when all the premises are wanting.

I have spoken of Goltz, and I must add that this author, who now adopts nearly completely my explanation of some facts, holds that tactile sensibility is not completely wanting after decortication of the excitable area, but that it becomes much enfeebled. I will not enter here on the discussion of this question, which has for us only a secondary interest, and cannot change our explanation of the symptoms. Even though a trifling remnant of tactile sensibility still remains, it is not sufficient to provoke a motor reaction in the ordinary state of the animal.[3]

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[1] See the outline figure of an ataxic cat in my paper in Pflüger's 'Archiv,' 1883, p. 275, Fig. 2.

[2] Pflüger's 'Archiv,' 1883, p. 264 et seq.

[3] Ibid. vol. xxxiv. p. 465.

Already in my paper of 1876 in the 'Rivista di Frenatria,' I remarked that a dog under the influence of strychnia, seems to be nearly equally excitable to tactile impressions on both sides, though on one side he has been deprived of the cortical excitable area. It is true he is excitable, but does he receive from both sides well-localised impressions? Some facts I have witnessed lead me to doubt that he knows that he has been touched on the anæsthetic side.

All I have said till now relates to full-grown or nearly full-grown animals. Is it the same for very young dogs? New experiments must be made on this subject. But I must add that in two instances, among more than 200 experiments, the state I suppose to exist in new-born animals was still existing in the latter part of the first year. And this *on one side only.* The other side of the brain showed the ordinary effect of decortication.[1]

All these facts, and those observed and analysed by other physiologists, justify the conclusions:

(1.) That the direct and only effect of the ablation of the excitable area in fully grown dogs and many other animals is tactile anæsthesia as observed by me, and insensibility for cold as first observed by Herzen.

(2.) That this effect persists without diminution as long as the animals live or can be observed.

(3.) That the simple and the co-ordinated muscular movements, or muscular innervations, are not involved so as to become either feeble or paralysed.

(4.) That some defect or some hesitation in the ordinary movements may arise in variable intensity through want of tactile sensation.

Such a state cannot be called paralytic, as Prof. Horsley would still call it. Seeking a name for it, the expression *Ataxy* presented itself very obviously. If I was wrong in adopting it (and Herzen followed me in that course, and Wernicke seems to approve of it), Prof. Horsley may propose a better one, and I shall accept it.

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[1] See for one of these cases the paper of Herzen and Löwenthal, in one of the forthcoming numbers of the 'Revue Zoologique Suisse.' The other is still living in my laboratory, and will eventually be examined post-mortem by me or my friend, Prof. N. Löwenthal.

But in the meanwhile it seems to me that we must preserve it, unless we wish to create a new one. Thus the position of the toes remains for us the best and most expressive sign of ataxy. And a dog which has lost the excitable area is not only ataxic, but appears to me, in the words of Homer :

πόλλον ἀτάξιος ἄλλων.

I have endeavoured to find somewhere on the surface of the cortex a true motor centre. I have not found one. Even after a complete section of the internal capsule of one side, there exists no motor paralysis.

Influence of the Posterior Columns of the Spinal Cord on the Excitable Area of the Brain.

We see from the preceding exposition that, from the beginning of my experiments, the analysis of the facts led me to the conclusion, that the pretended motor centre does not exist in dogs, and that it must really be called a sensory centre, if it is a centre at all, and not, as I believe, the most central patch of afferent channels.

A new experiment I made some years ago led me to the conclusion, that what I have considered to be an analogy between the effects of decortication of the brain, and of division of the posterior columns of the spinal cord, is more than a mere analogy, that it is identity. The fifth day after the division of the posterior columns of the cord, the excitable area of the brain loses its influence, and no longer gives rise to reflex actions in the parts of the body posterior to the division of the posterior column.

Prof. Horsley, without being acquainted with our method of experimenting, believes that he has repeated our experiments. His results were in general negative, or nearly so. I must even regard as negative the result of the experiments in which the Professor found my result "marked" or "very marked," because, in order to confirm my experiment, I must demand that the result shall be confirmative in a quite *absolute* manner. It was not so, and this is not surprising. It would have been much more surprising, had he by his method been able to confirm my results.

I know only one method for making the resection of a part of the posterior columns without damaging the rest of the spinal cord. It is the method indicated in my text-book,[1] and to which I have always returned after having examined other apparently simpler and easier methods. By it all pressure is avoided on the so-called motor tract, when we make the first incision very obliquely and very incomplete. That even a slight pressure on the (true) motor tract is very fatal in the operations on the spinal cord, is not, as Prof. Horsley believes, a new discovery of his own, but has been largely studied and set forth in my text-book, p. 278. I had given there a list of the different authors who, up to that period (1858) had been led into error by the effects of unintentional pressure on the motor elements. The resistance of these latter to pressure is less than the resistance of the sensory elements. I believe that, by this remark, I have given the key for the explanation of some paradoxical facts met with in clinical observation.

Prof. Horsley kills his dogs about the fifth day, and excites the brain with an ordinary Du-Bois Reymond's coil worked with one Daniell's cell, and in one case with two. I think that currents of so high a tension and so short duration as induction currents, are not the best nor the surest means for exciting a large parenchymatous organ like the brain. Instantaneous currents must have a very great intensity to produce a reflex movement; and the so-called "motor centres" really act by a reflex process. The intensity of the current is a cause of extensive derivations, and so we are never sure to localise exactly an induction excitation in an organ the form of which is not linear. In another paper on a recently published case of Baldi, on the brain, I shall show that all his experiments have been lost by derivation into deeper parts of the brain, and even into the roots of the fifth nerve. It is for these reasons, that in experiments on the excitable area, I do not trust induction currents, and I have always made the decisive experiments with a simple galvanic battery with a rheocord. The makes and breaks are made with the hand by means of a Morse interruptor. And indeed, in my own paper on the experiment, which forms the object of Prof. Horsley's criticism,[1] and which the Professor seems to have overlooked, I recommend for the excitation the use of 4 to 16 Leclanché cells; and only exceptionally make use of a *single* induction opening current (the hammer of the instrument being fixed) as a corroborative experiment when it gives no contraction, and as an experiment of no account, when I had an effect which could not be obtained by the battery current.

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[1] 'Physiology of the Nervous System,' p. 242. See also 'Comptes Rendus,' 1854, p. 926.

We see that Prof. Horsley has been drawn into error when he says (*l. c.*, p. 47) that my method of experimentation, as far as he knows it, exists simply in exposing and dividing the posterior column on one side. I have always made, not the *division* but the *resection* of the two posterior columns, or of one. I never excited with an inductive current rapidly interrupted by vibrations of the hammer. I made the operation on various parts of the spinal cord, always by the method indicated in my book. I made and broke the current (even the induction current) with the hand,[2] never taking it too strong. I have much insisted on this method, which I think must give to Prof. Horsley, if he will really repeat my experiments, the same result as I have obtained in my own numerous experiments.

And this result is not, as Prof. Horsley supposes that I "would have him to believe," that the so-called motor centre becomes unexcitable, which are in strict reflex relation with the muscular groups, separated from the "centre" by the resection of the posterior columns. It is quite in harmony with my results, that after the division of the posterior columns within the fourth vertebra, he should still meet with what he calls flickerings in the trapezius, and even in the face, produced by excitation of these pretended centres.

But Prof. Horsley believes that *no* excitability is lost, but that his "centre" can no longer *transmit* its excitation, because a degeneration of the pyramidal, or, as he says, motor tracts, puts a block against the peripheral extension of the innervation. Let us now examine this opinion.

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[1] Pflüger's 'Archiv,' xxx. p. 257 et seq.

[2] I must add, that during the last few weeks I have found a method of using a modification of the Clarke magneto-electric machine with mechanical interruption, which can be used for experiments on the brain, without exposing us to errors produced by derivation of the current.

Can the Pyramidal Tract of the Lateral Columns be regarded as Motor?

When Prof. Horsley first published his objections against my experiments and claimed them to be produced by a lesion of the motor tract, I did not understand what he meant by this expression of "motor tract," and with its unintentional lesion. The microscopical examination of transverse sections of my spinal cords, showed no accidental degeneration. That I made this examination long before Prof. Horsley could draw my attention to it, is proved by what I said in Pflüger's 'Archiv,' 1883, p. 264, where I concluded that the operation on the posterior columns did not produce a degeneration, neither total nor partial, of the pyramidal tract. But I must add here, that only a small part of the posterior grey horns was filled with white corpuscles, the nerve fibres being intact; and there was not even in these horns any trace of descending or ascending degeneration.

But why did I not understand the meaning of Prof. Horsley? I believe I have established, in my book of 1858 (and in the previous communication published by the Natural Society of Berne and the Institute of Paris), the first lines of an experimental physiology of the spinal cord. My researches have held their ground against many contrary results, which since that time have been announced, soon to disappear. So I was accustomed to my own terminology, and I did not acknowledge the existence of a special motor tract. In Prof. Horsley's last paper I find the explanation of his meaning, and it is on this occasion that he sets forth his discovery of the influence of pressure on the motor, or more properly *kinesodic,* elements of the spinal cord we have already spoken of. He had, by his method of operating, produced a more or less extended degeneration of the undivided, but compressed, lateral tract, and he erroneously supposes that I must have done the same, and that this assumed degeneration of the pyramidal tract of the lateral columns *can,* or as he thinks *must,* cause an interruption of the transmission of movement from the brain to the limbs.

The degeneration he produces enters into the category of what the French school has hitherto called a *sclerosis.* But I have proved long ago that several forms of this so-called sclerosis do not destroy the cylinder-axis in the nerve-fibres, but are an expression of what I have called an Atelectasis of the nervous tracts.[1] Westphal's criticism of my view could not shake, but rather confirmed it, leading me to new confirmatory researches. In my paper on Atelectasis (1880), I arrived at the conclusion that, in many cases, the so-called sclerosis cannot interrupt the transmission of nervous influence—a conclusion which I applied at that period to the cases of posterior sclerosis described by Friedreich, Gowers, Pick, and others, where the sensibility was not at all, or scarcely, disturbed. Last year a new confirmation of my views arose from the discovery made in Paris, that a certain number of cases of sclerosis of the pyramidal tract presents no secondary descending degeneration. But a true block in the transmission, a transversal interruption, is always followed by descending degeneration of the pyramidal tract. Hence these cases of sclerosis do not produce a block. And this conclusion has been reached even by some neurologists, who formerly were my adversaries on the question of Atelectasis.

Degeneration of the pyramidal tracts is not produced in the experiments made according to my old method. I may regard as non-existing some experiments which I made after another method, with the "microtome" of Strauss-Durckheim,[2] in which the lateral tract was really injured.

But I can prove that this degeneration of inflammation of the pyramidal tract, even if we suppose with Prof. Horsley, and on the evidence of microscopical examination, that it has been produced in *all* my experiments on the posterior columns, *cannot be made responsible for the partial excitability I found in the so-called motor centres of the cortex.*

Prof. Horsley remarks, p. 47 of his paper, that "English neurologists, at any rate, will be interested to learn (*vide* 'Lancet,' 20th February, 1866) that Prof. Schiff is about to combat the general opinion, that the crossed lateral tract is motor." They will be still more interested to learn that I am not about to combat, but I have combated for a long time this general opinion (in the case of dogs and cats), by a series of very clear and non-equivocal experiments.

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[1] See my paper on "Atelectasis Medullæ Spinalis" in Pflüger's 'Archiv,' xxi. p. 238, and also the criticism of Westphal in his 'Archiv.'

[2] See his 'Traité d'Anatomie comparative;' Paris, 1842, tome i. planche iv. fig. 59.

My old opinion, resulting from a large number of experiments, that the lateral column does not contain fibres the section of which produces motor paralysis—an opinion which has since been fully confirmed by other experiments—may have been regarded as insufficient, as having been formed in a period (1850–58) in which no special attention has been paid to some special reflexes, which are distinguished as voluntary and unintentional, from these movements which depend upon a general and pre-reformed mechanism.

Pflüger's 'Archiv,' vol. xxx. (1883, pp. 243–248) contains a series of experiments with complete section of the pyramidal and partial section of the cerebellar tract (on one side), which prove that not only the general movements, but even the intentional ones (such as a dog giving the paw, holding the bone with both anterior legs, a cat climbing and catching objects with the limb on the operated side) are preserved, though there was not only interruption, but also a complete descending degeneration of Horsley's so-called motor tract. The degeneration, though it did not extend down to the origin of the roots of the sciatic nerve, but stopped somewhat higher, was *more complete* than that generally visible after the extirpation of the excitable area of the brain. These degenerations, and their differences in extent after injury of the brain or transverse section of the pyramidal tract of the spinal marrow, have been studied in the same animals by Prof. Löwenthal.[1]

Within the last few months these facts have been independently confirmed by Herzen and Löwenthal in a young cat, in which they had cut the pyramidal tract of the lateral column. This column showed very extensive degeneration.[2] Herzen has very well described the voluntary movements, though the cat was not kept alive long enough to allow them to be fully re-established with reference to their strength.

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[1] See his communication in Pflüger's 'Archiv,' xxxi. p. 350, with the accompanying Table IV.

[2] See 'Archives de Physiologie,' April 1886, p. 20, and the plates.

Though the full energy was still wanting, it is clear that the movements were all present.

We cannot suppose that, in these animals, the preserved *direct* pyramidal tract in the anterior columns has in some degree compensated for the destruction of the lateral crossed tract, because we know now that, at least in dogs and cats, there is nothing of the nature of a direct pyramidal tract.

The experiments of mine I have just mentioned are not decisive. At the period I made them I was using the microtome of Strauss,[1] which must be introduced by stretching, and so I produced indeed extensive lateral compressions and degenerations outside and below the divided parts. Microscopical examination of the hardened marrow[2] revealed afterwards that these errors were due to the same conditions, which Prof. Horsley wrongly supposes complicated my sections of the posterior columns. And so I produced some symptoms, which at that time I attributed to the absence of the transmission in the pyramidal tract, but which really depend upon a concomitant alteration of the posterior columns, which has been found later in the hardened parts. But even before the microscopical examination I expressed my surprise (*l. c.*, p. 243) that what I took for the effect of section of the pyramidal tracts, resembled so much the symptoms of resection of the posterior columns, and that an intentional transversal section of the pyramidal and the posterior columns made together, and simultaneously, produces no other effect than section of the posterior column alone.

So I committed at that time the error recently imputed to me by Prof. Horsley, but quite in an inverse sense; I attributed too much to the pyramidal tracts, the section of which, as we know by later and more correct experiments, produces no visible symptoms, not visible at least with our methods of research.

But even in the former vicious experiments all the movements were present; voluntary, isolated movements were not wanting, though they were modified by insensibility, and so true to the principle and the method indicated in my text-book of 1885, p. 231. I can give credit even to those former experiments establishing *that a complete degeneration of the pyramidal tract, and of all the posterior half of the lateral column, does not produce in our animals loss or diminution, either in the voluntary and isolated, or in the general movements of the parts behind the section and behind the beginning of the degeneration.*

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[1] See Pflüger's 'Archiv,' *l. c.* p. 241.

[2] See Löwenthal, 'Des Dégénérations secondaires de la Moëlle Épinière.' Thèse; Genève, 1885.

There was still one question open, and it is for this purpose that I began two years ago a new series of experiments, conducted according to my old trustworthy method. Does excitation of the excitable area of the brain, opposite to a complete degeneration of the pyramidal tract, still produce movements of the limbs of the degenerated side? Some weeks or some months after the section of the posterior part of the lateral column (pyramidal tracts) of one side, I began the experiment by exciting the brain with a battery current, and in some cases a single induction shock. The (reflex) movements in the limb, which are normally seen by stimulation of the excitable area, were present by excitation on *both* sides of the brain. The section of the pyramidal tract did not hinder or enfeeble the effect observed in the experiments of Hitzig and Fritsch. The experiments of Ferrier (with quickly interrupted induction currents) probably do not fail in these cases; but if I had excited the brain immediately after having cut the pyramidal tract, the stimulation would have remained without result. It is essential to let the traumatic irritation pass off before testing.

When in a dog or a cat, in which one pyramidal tract has been divided and has long been degenerated (from the cervical region downwards), we make a deep and extensive extirpation of the so-called "motor-centres" of the opposite side, we can before traumatic equilibrium is reached, *i. e.* in the first weeks, observe all the symptoms which have been regarded as proving the absence of intentional movements on the side opposite to the operation, and the characteristic anæsthesia is present and maintains itself indefinitely. Hence all these symptoms produced after the second operation cannot be dependent upon the existence of the pyramidal tract, in which our previous operation had caused to degenerate.

It is evident that the suppositions of Prof. Horsley, and his mode of viewing my experiments on the posterior columns, are quite erroneous. They cannot be admitted, even if it were true that I had compressed what he calls the motor tract. And they must be rejected *à fortiori*, because I had not compressed this tract.

The crossed pyramidal tract finds its nutritive centre in the neighbourhood or within the excitable area, but there is no known functional relation between this area and the lateral columns of the spinal cord.

When in 1853, after Waller's and my own experiments, I first proposed and defended the difference between the nutritive and the functional centres, there was among anatomists and physiologists much discussion and contention about this subject. Here we find another and a very striking example of this difference. This example must hold good, at least till we know something of the functions of these pyramidal tracts. It seems, after an observation of Beck, that they can be cut even in man without the loss of voluntary movement.

One of my pupils, Mr. Schopen, is now about to publish some observations of injury of the anterior part of the brain on one side accompanied by anæsthesia (for tactile excitations). In one of these cases (and in two similar I observed some years ago) the secondary degeneration was wanting in the pyramidal tracts. In two other cases there was degeneration of equal extension and intensity in the two crossed pyramidal tracts,[1] that is, on *both* sides, and notwithstanding anæsthesia and its allied symptoms were present only on the side opposite to the cerebral injury. The extirpation in these cases was not in the posterior limb of the sigmoid gyrus, but between the sulcus cruciatus and the frontal (supra-orbital) lobe. Without entering here into particulars, it is evident that such cases offer new proofs, that the functional and the nutritive disturbances are independent one of the other.

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[1] The extension of the degenerations and the number of preserved nerve-fibres have been determined with the polarising apparatus. Two determinations were made for each section, one simply with crossed Nicol's prisms and one with the aid of a selenite film.

Functions of the Posterior Columns.

The preceding exposition shows, that the opinion of Prof. Horsley is not admissible, when he says that all we have hitherto attributed to the posterior columns may be due to a concomitant lesion of the lateral columns. He states, that in the cases of section of the posterior columns in which only a very slight lesion was found in the lateral, he could not verify the tactile insensibility found by me, and he did not investigate the insensibility for cold found by Herzen. That he could not verify the loss of tactile sensibility in all cases may depend either on the same causes which hindered Ferrier—to whom Prof. Horsley refers—from finding the insensibility after an injury of the excitable area of the brain, or it may depend on the method of dividing the columns. This division, as I have still to show, was imperfect in the beginning of his observations, and very slowly spread by traumatic inflammation to a perfect destruction of the column. If it is so, an examination during the first, the second, even the third day, might have given him an imperfect result.

It is probable that if Prof. Horsley had examined the dogs of this series as we examine the animals after decortication of the excitable area of the brain, he would have convinced himself that all local disturbances after these two operations are absolutely the same. I say *local* disturbances, and I should have added persistent, because the first traumatic disturbances are somewhat different. Besides, the brain is in relation with the entire half of the body (though I have hitherto been unable to produce a cerebral insensibility of the inner surface of the ear). The posterior column acts only on the parts below the operation.

Instead of this, Prof. Horsley contents himself with three methods of examination, which are very fallacious. Fallacious in the sense that their application leads into error, when they are not exactly limited and graduated as to act only on tactile sensibility and not on the other sensibilities. It is so, for instance, with reference to the "clip" Prof. Horsley mentions (*l.c.*, p. 49). It is true that very often I make use of a clip, as Goltz did before me, but I employ it rather for demonstration than for researches. I have three pairs of clips of different strength and resistance. First, the strongest is applied successively on both sides of the body. Generally sensation is indicated on both sides. But I am not yet warranted thereby to conclude, that tactile sensibility equally exists on both sides, because the clip may act by pressure. I take a weaker one, and often a weaker still, and so I become enabled to conclude, that there exists a certain degree of contact well perceived on one side and not on the other side of the body.

The same precaution must be applied, in a still higher degree, when you touch the skin with a needle, or when you allow an object to fall on a sensitive surface, even when this object is simple water (see *l. c.*, p. 49). So it is evident that the mere enumeration of the three methods of testing used by Prof. Horsley do not warrant the exactness of his result.

These methods are not sufficient for making me believe, that Prof. Horsley has found three exceptions to a general rule, which have held good till now for many years, in very numerous experiments, without a single exception, and which has been confirmed in many species of mammals.

The ascending degeneration after resection of the posterior columns must go in some manner as far as the cortex, but I never pretended, as Prof. Horsley would have his readers believe, that it goes as far as the ganglion cells producing some visible alteration of these parts. I can add, after Löwenthal's examination of the brain of some of my dogs, that the visible degeneration does not reach beyond the pons Varolii, but it can be partly followed as far as the cerebellum.

If I have several times insisted on the fact, that I make not a section, but a *resection* of a piece of the posterior columns, I do not imply that this difference is of any importance for the result, but the difference is capital for the method. We can never make a complete section, and be sure of sparing the neighbouring parts; nay, we may be certain of damaging them.

How Professor Horsley's Results were obtained.

If it is allowable to put forth any such supposition, I think that these results are due in part to the method of operating, and partly to the mode of the excitation. Prof. Horsley makes (*l. c.*, p. 48) in a posterior column a small punctured incision, with a sharp-pointed tenotomy knife. It can scarcely be supposed that there exists a form of tenotomy knife which, with a simple puncture, divides all the posterior column, and nothing but this column. And though Horsley's figures generally show the lesion to be very exactly limited to the column, after their inspection I must assume that he made a puncture which did divide only a part, a stratum of the column, and that the rest was compressed and did disappear successively during the five days the animal was living. So a large part of the column found damaged at death may have been destroyed only three, or even two days previously. My experiments[1] have proved that one or two days after the resection of the posterior columns the brain remains excitable. In a more recent experiment, which will be communicated by Schopen, it remained still excitable four days after the resection of one posterior column. It must be partially excitable when a part of the posterior column has been divided only for two or three days.[2] So the imperfect movements perceived by Horsley after his excitation of the brain can be understood.

So we can understand how the fissure of the wound contained débris of nerve-fibres which are not present in a sharp, well-cut wound. The sides of the wound-cavity, says Prof. Horsley, "although so extremely small, showed clearly that there was some degree of tension in it." This can be understood to mean, that the walls of the fissure showed some irregularity. This must be so, even in the cases when an incomplete fissure increases by parietal inflammation. Had Prof. Horsley kept his dogs some days more, his results probably would have been more complete.

I have already spoken of the method of excitation. The use of induction currents has produced so many errors in the physiology of the brain, and more than elsewhere in the hands of English physiologists, that it is high time to give up the use of the induction coil and to return to the galvanic battery with a rheocord, which allows us to regulate the necessary variations in the strength, and in the duration of the current.

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[1] See Pflüger's 'Archiv,' vol. xxx. p. 260.

[2] See the experiment of Herzen and Löwenthal on a cat. 'Archives de Physiologie,' April 1886.

In writing the foregoing pages, it has been my aim to present the English reader with a sketch of the actual position of the question, and to give him some indications which he will seek in vain in the annual reports on the progress of physiology. I must return my best thanks to Professor Horsley for his zeal, and the interest he has shown with reference to our recent researches. I hope that this paper will engage him to undertake, on a new basis, a fresh series of experimental researches, on a subject which fully deserves the attention of English physiologists and neurologists.

A FURTHER AND FINAL CRITICISM OF PROF. SCHIFF'S EXPERIMENTAL DEMONSTRATION OF THE RELATION WHICH HE BELIEVES TO EXIST BETWEEN THE POSTERIOR COLUMNS OF THE SPINAL CORD AND THE EXCITABLE AREA OF THE CORTEX.

(*An Answer to* Prof. SCHIFF'S "*Reply*" *in this issue.*)

BY VICTOR HORSLEY, B.S., F.R.S.,

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IN 'BRAIN,' April 1886, I published a paper descriptive of seven experiments I had performed, to test the value of an observation by Prof. Schiff, which was considered by that author to wholly negative the view, that the excitable area of the cortex could be considered to be motor in function.

In criticism of my paper, and in answer to my statements, Prof. Schiff wrote the "Reply" which precedes this paper. I feel therefore bound to again draw attention to my repetition of his experiments, and the more so since, in his "Reply," he has made certain definite charges against me. In my paper I made a brief criticism of Prof. Schiff's general views, in order to make clear the purport of his experiment which I repeated. I then described my work in full detail, showing that I obtained Prof. Schiff's result perfectly in some instances, but not in others.

Finally, I showed that this discrepancy was due to the fact, that Prof. Schiff's positive result occurred in consequence of an experimental accident, and that therefore his deductions from the same were unsound.

To this criticism Prof. Schiff has returned the somewhat elaborate answer which precedes this paper. In attempting to meet my criticism of his experiment, he makes some very definite statements, of which, however unwillingly, I am compelled to take notice, and that in very full detail. Prof. Schiff's charges against me may be conveniently grouped under three headings.

I. Criticism of his views of the function of the excitable area of the cortex.

II. His experimental method.

III. English methods of experimentation on the central nervous system.

As Prof. Schiff's "Reply" is rather diffuse, I am compelled to answer him, at the risk of incompleteness, by the tedious method of collecting together all his objections under the above headings, and disposing of them one by one. And though I shall show that these objections, as such, have not really the force which Prof. Schiff's dialectical skill appears to give them, and that it is therefore unprofitable to discuss them further, I think in the interest of neurology, and especially English neurology, that it is highly advisable such statements as these of Prof. Schiff should be most thoroughly examined; for although controversy is not the best, and certainly not the most pleasant method of arriving at truth, still there can be no doubt that, by its means, facts and theories are dragged out into the light, and are thus either rescued from obscurity, to the great profit of science, or else justly condemned to oblivion.

In the establishment of a new science on the sound basis of truth, the free cosmopolitan interchange of facts and views is of the highest importance. Especially is this the case with the science of Cerebral Physiology, which, practically speaking, is not much more than a decade old; therefore, although we may differ from Prof. Schiff in our estimate of the value of the facts on which he bases his views, still, it seems to me, we are under a considerable obligation to him for having presented the question, with indefatigable energy, to us in a different light.

As neither have I the time to devote to discussion of the general ideas prevailing on the subject of cerebral physiology, nor is this the opportunity for doing so, I shall proceed to examine the objections contained in his "Reply," as exhaustively as I possibly can in this paper, since I shall be unable, for the reasons just stated, to discuss the matter further.

I.—Prof. Schiff's objection to my criticism of his views.

Prof. Schiff says that I should not have contented myself with criticising merely his opinions as set forth by Dr. Huggard, but that I ought to have reviewed the whole of his own previous papers on the subject. Indeed, he seems to consider that I have in consequence misunderstood and misrepresented him. Whether I have misrepresented him or not, can only be determined by a comparison of what he has written in Pflüger's 'Archiv' (Band xxx.) and elsewhere, with the following excerpt from my paper, which states what I understand to embody his view.

"C. Prof. Schiff's View.—Prof. Schiff states that he has traced (physiologically) the afferent fibres (of the posterior column of the cord) directly to the excitable portion of the cortex, which he therefore regards as being the[1] sensory perceptive centre for tactile sensibility. From this sensory perceptive area he believes that fibres go down to the motor centres, which are situated he knows not where."[2]

Since this is nothing more than a comprehensive statement, in words, of the opinions expressed in his paper in Pflüger's 'Archiv,' Bd. xxx., I feel the charges of being ignorant of, and of ignoring, his previous writings to be as much without justification as they are paradoxical.

If my reading of the same was wrong, Prof. Schiff should have directly met my statement with a counter-statement, which I cannot find that he has anywhere done in his "Reply."

I shall therefore, in the absence of any correction by him, continue to consider that what I have said is a faithful exposition of his views.

DIGRESSION.

After preferring the above charge against me in the first page of his "Reply" (repeated at intervals farther on), he devotes nearly half of his remaining space to reasserting the worth of his experiments on extirpation of the motor cortex in dogs, and to restating his deductions therefrom.

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[1] I should perhaps have said, *part of* the sensory perceptive centre, for it is difficult to make out from his Schema, figured on p. 267, *l.c.*, that he really thinks the cortex cerebri to have any function at all.

[2] 'BRAIN,' April 1886, p. 43.

Now in my paper,[1] I particularly state that, for reasons then given, I would not discuss this experiment, and I only alluded to it, as I before explained, in order to make the object of my communication clear, viz. the criticism of Prof. Schiff's method of investigating the relation between the posterior columns of the spinal cord to the excitable area. Neither do I intend to discuss now this enormously wide subject of the representation of sensation (tactile, motor, &c.) in the excitable cortex, since I consider that the question is entirely an open one, and I am therefore unprepared either to deny or acknowledge the validity of Prof. Schiff's views on this point.

Moreover, it is not germane to the matter in dispute, viz. Prof. Schiff's method of experiment on the spinal cord.

In justice to myself, however, I cannot pass unnoticed a passage in Prof. Schiff's "Reply" by which he apparently seeks to avoid the responsibility of his whole theory relating to sensory paths and the cortex. The passage referred to is to be found on p. 28, and runs as follows:—"The ascending degeneration after reaction of the posterior columns *must go in some manner as far as the cortex*;[2] but I never pretended, as Prof. Horsley would have his readers believe, that it goes as far as the ganglion cells producing some visible alteration of these parts."

Now if the latter half of this sentence means anything at all, it says that some wonderful, and certainly never before heard of, disappearance of excitability in the ganglion corpuscles of the excitable area of the cortex follows ascending degeneration of the posterior columns. Although the said degeneration is supposed never to reach the corpuscles themselves, nevertheless it is at the same time supposed to "go in some manner as far as the cortex." It would be difficult to imagine a degeneration getting to the cortex, except as a degenerative process. But even putting the most charitable interpretation on Prof. Schiff's statement, in the interest of scientific fact I must quote again from the paper he seems to think I am not sufficiently familiar with, viz. Pflüger's 'Archiv,' Bd. xxx. On p. 259 he says, after describing the original experiment, my objection to which, in 'BRAIN,' April 1886, has caused the present controversy:—"Neu an der Sache ist, abgesehen von der durch sie gebotenen schlagenden Demonstration, nur, dass die aufsteigende Degeneration, deren obere Grenze man nicht kannte, bis zur Gehirnrinde hinansteigt. Dies soll auch bald anatomisch untersucht werden."

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[1] 'BRAIN,' April 1886, p. 46.

[2] The *italics* are mine.—V. H.

I need hardly ask what statement can be couched in more definite terms than this, or with more reliance upon the experiment referred to. It perfectly justified me in stating that Prof. Schiff believed the degeneration to reach (hinansteigen) the cortex; but of course I could not know that Prof. Schiff would suddenly repudiate in 1886 what he wrote in 1883. It is an instructive comment, however, on the question, that when the "anatomische Untersuchung," Prof. Schiff mentions, was made by Prof. Löwenthal (see p. 13 of his "Reply"), all the force was taken out of the above-quoted ex cathedrâ statement.

In passing to the next point, viz. Prof. Schiff's views of the representation of movement, I cannot help expressing my regret, that he should require of his readers to grant such very liberal elasticity to the meaning of his terms, as it makes discussion exceedingly difficult, and indeed well-nigh impossible.

Intimately connected, however, with the real point at issue, namely the relation of the posterior columns of the cord to the excitable area of the cortex, is the question of representation of movement in the excitable cortex, and the nature and anatomical position of the paths of nerve impulses from that portion of the brain to the spinal-cord centres. Prof. Schiff has, in previous papers, dealt with this problem at considerable length, and in his "Reply" to my criticism of his experiment on the spinal cord, has reiterated his views in plain language; so that, if I hereafter prove some of his statements to be inadequate, at least he cannot charge me with not comprehending his meaning.

I must enter here at length into a consideration of Prof. Schiff's views concerning the position of motor centres and paths, since it is by clearly keeping all the facts in view that his attack on my repetitions of his experiments can be immediately seen to be in itself perfectly contradictory.

In the first place, whereas most neurologists will admit, that Dr. Ferrier's experiments have proved that the representation of various movements is localised in certain portions of the excitable area of the cortex, Prof. Schiff places himself on exactly the opposite side, by denying that there is any motor centre in the cortex at all, and by asserting that, in his opinion, no movement whatever is represented in the cortex as such. The cerebral motor centres he apparently believes to be situated somewhere in the base of the brain. Further, Prof. Schiff believes that the fibres which make up the direct pyramidal tract, and those theoretical ones which connect the centres in the excitable cortex with his theoretical motor centres in the base of the brain, are not *motor* in function, but *kinesodic*.

With the second theoretical group of fibres we have nothing to do. Now with regard to the pyramidal tract, Prof. Schiff denies that this is a specially *motor* tract, but admits it to be *kinesodic*. Most neurologists, perhaps, would not distinguish between these terms *motor* and *kinesodic;* but in the 'Comptes Rendus,' 1854, p. 930, third line from the top, he says " . . . . . et il m'a paru qu'il fallait les distinguer des fibres sensitives par un nom nouveau, et je propose de les appeler fibres *esthésodiques* (ὁδὸς, path), et je prouverai plus tard qu'il y a, dans la même substance, des fibres très analogues pour les mouvements que j'appellerai *kinésodiques*."

Whether he calls the pyramidal tract motor or kinesodic matters nothing in the present question, for my simple contention was that it conveyed *efferent* impulses from the " motor " cortex, and consequently that a block in it would explain the result of the experiment.

At any rate, it is perfectly clear that Prof. Schiff refuses to call the pyramidal tract motor. What then, it may be asked, does he consider to be its function, and what is its relation to the excitable area of the cortex ? Fortunately, he has summed up his ideas on this subject in one passage (Pflüger's 'Archiv,' Bd. xxx., p. 249, bottom line):

"Die Hinterstränge bis ins Hirn hinein bilden die Legislative, die Türck'schen Bündel erwecken die Exekutive."

From this it is quite clear that he believes the posterior columns carry impressions into the brain, and that the pyramidal tract carries impulses from the brain which set the muscles in action. But the latter of these beliefs is exactly what I have contended to be the case; and the only difference between myself and Prof. Schiff lies in the fact, that I prefer to call the pyramidal tract *motor*, while he considers that the only term applicable under the circumstances is *kinesodic*.

Let us now follow Prof. Schiff a little further, and seek from his writings to discover what he believes to be the *functional* relation between the motor cortex and the pyramidal tract. If we simply trust to what I have just quoted from Pflüger's 'Archiv,' it would seem superfluous to do more than accept his dictum, that the pyramidal tract "erweckt die Exekutive," if he had not in most emphatic language in his "Reply" preceding this paper declared, that (p. 25) "The crossed pyramidal tract finds its nutritive centre in the neighbourhood or within the excitable area, but there is no known functional relation between this area and the lateral columns of the spinal cord," *i.e.* the pyramidal tract. Now if there is no functional relation, how comes it to "erwecken die Exekutive"? But a still more pertinent fact tending to elucidate this contradiction in Prof. Schiff's statements is one of his own experiments, upon which he based the idea of the pyramidal tract being executive to the motor area. The experiment I would refer to is described as follows (Pflüger's 'Archiv,' Bd. xxx. pp. 248, 249, commencing eight lines from bottom of p. 248):

"Einem gehörig ätherisirten Hunde wurde z. B. die *linke* vordere Hirnoberfläche und das oberste Lendenmark blossgelegt. . . . (unimportant details) . . Eine Kette von 4 Elem. kleiner Leclanché erweckte von der Gegend des Gyrus sigmoideus aus starke Zuckung entweder in beiden Extremitäten der rechten Seite, oder weiter oben nur im Hinterfuss. Es wurde auch eine Stelle bestimmt, die nur auf die vordere

Extremität und den Kopf wirkte. Jetzt wurde rasch mit dem Mikrotom der rechte Türcksche Bündel (mit seinen unvermeidlichen Begleitern) durchschnitten ohne Blutung. [The pyramidal tract was thus divided opposite the first lumbar vertebra.] *Keine Hirnreizung*[1] mit 4 Elementen erregte mehr Bewegung im rechten Hinterfuss, wohl aber in der vorder Extremität, u. s. w."

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(Continuation, p. 249, line 15 from bottom.)

"Durchtrennung der Bündel am obere Halsmark. *Nach mehreren Tagen*[1] Aetherisation, Blosslegung des Gyrus sigmoideus und seiner Umgebung. Vergeblich wurde sogleich und bei Nachlass der Betäubung nach einem Hirnfelde gesucht, dessen Reizung selbst mit starken (Ketten- oder Induktions-) Strömen Hitzig'sche Zuckungen gegeben hätte. Selbst Einbohren der Elektronadeln blieb erfolglos.

"Endlich wurde, um den Verdacht einer möglichen Wiederherstellung auszuschliessen, die Hirnreizung bei Hunden vorgenommen, denen schon vor Wochen der Bündel [*i.e.* the pyramidal tract] auf einer oder auf beiden Seiten durchtrennt worden. Der Erfolg war wesentlich derselbe und auch hier der Höhe der Durchschneidung entsprechend verschieden."

Now I have quoted this experiment at full length because it expresses a fact, and not a theory; a fact, too, upon which the whole of my criticism of Professor Schiff's experimental investigation of the relation between the posterior columns of the cord and the excitable area of the cortex depends. In the first part of the just-quoted experiment, Prof. Schiff demonstrates irrefragably that, if the pyramidal tract be divided, the constant result is absolute blockage in the path of the nerve-energy from the cortex to the spinal centres, that energy being evoked by stimulating the cortex. He thus *proves* that the pyramidal tract is the path of efferent impulses from the excitable area. It is therefore perfectly incomprehensible to me how he can assert, as he has done (p. 25), in his "Reply" to me, that there is no functional relation between the excitable area and the pyramidal tract. His statements of 1883 and 1886 are absolutely contradictory.

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[1] The *italics* are mine.—V. H.

I prefer to believe the experimental fact of 1883, and not the controversial theory of 1886.

Further, it is extraordinary to my mind that Prof. Schiff, in his anxiety to contest the propriety of calling the pyramidal tract motor, and desiring us to adopt his expression kinesodic, should thus ignore the statements and experiments contained in his original writings, to the study of which he so frequently refers me. In his "Reply" it will be seen that he elaborates at great length the statement, that the motor disturbances (for his sake we will not call them paralyses) produced by section of the pyramidal tract in the dog are not permanent; while he wholly ignores the well-known fact, that in the dog, fibres pass down both sides of the cord from each hemisphere; the recovery of power, when only one tract in the cord is injured, being thus easily explained. Besides, we do not know what functional relation the fibres in the rest of the antero-lateral column bear to the "motor" cortex; and finally, the question whether the effects of section are permanent or not, has nothing to do with the point under discussion—viz. whether the pyramidal tract conveys efferent impulses or not.

As I must now leave this point, I trust it will be remembered that Prof. Schiff, as I have just shown, has himself demonstrated, that *damage to the pyramidal tract forms an almost absolute block to the passage of impulses from the excitable area of the cortex to the muscles.* It will be unnecessary to review at further length Prof. Schiff's views as to the situation of motor centres and paths in the brain and spinal cord, since I have quoted sufficiently from his writings to demonstrate that he has proved the pyramidal tract to be "efferent," and since he has not himself succeeded in discovering the situation of any motor centres in the brain.

II.—Prof. Schiff's objections to my criticism, and to my repetition of his experimental method.

This point, viz. the discussion of Prof. Schiff's experimental method of demonstrating the relation which he believes to exist between the posterior columns of the spinal cord and the opposite "motor" cortices respectively, is *the* source of disputation between us. I have, however, written the foregoing pages concerning Prof. Schiff's statements and theories regarding his view of the cerebral "motor" centres, since I consider that, in such an early and critical stage as that which cerebral physiology has arrived at, no pains should be spared to render perfectly clear the lines of thought which dictate the interpretation by others of experimental phenomena bearing on this subject.

I have said sufficient to show that Prof. Schiff believes the so-called "motor" cortex to be connected with the spinal centres by means of an efferent channel called the pyramidal tract, and further, that when this is injured, it is no longer possible to obtain movements by stimulating that "motor" cortex to which the injured pyramidal tract serves as a conductor of out-going impulses. Reference to the paper I published in 'BRAIN,' April, 1886, will show that I there demonstrated that occasionally, after section of the posterior columns of the cord, there ensued degeneration in the neighbouring pyramidal tract, close to the seat of operation. Again, that when the division of the posterior column was very exact and the wound healed at once, no degeneration occurred in the pyramidal tract. In the former series of cases, excitation of the opposite motor cortex was followed by the negative result, ascribed by Prof. Schiff to depression of function in that so-called motor area, due to the ascent of the degeneration (or degenerative influence?) process. Finally I showed that when, as in the second series of cases, there was no block in the pyramidal tract, this negative result of Prof. Schiff did not occur. From these facts Prof. Schiff seeks to dissent in two ways: first, by stating in fairly clear language (which I shall presently quote in full) that I have not obtained his result; and secondly, that my experimental method bristles with errors. These contentions I shall now rebut.

Professor Schiff's assertion, that I have not obtained his result, is introduced by him in the following words (p. 15): "Prof. Horsley, without being acquainted with our method of experimenting, believes he has repeated our experiments. His results were, in general, negative, or nearly so. I must even regard as negative the result of the experiments in which the Brown Professor found my result 'marked,' or 'very marked,' because, in order to confirm my experiment, I must demand that the result may be confirmative in a quite *absolute* manner. It was not so, and this is not surprising. It would have been much more surprising had he by his method been able to confirm my results."

I shall content myself with meeting this very distinct charge against my experiments, of incompleteness, and indeed inadequacy, by comparing his own description of his own experiment, word for word, with my description of my repetition of the same, and I think the *absolute* and confirmatory character of my observation will be "surprising" even to Prof. Schiff.

PROFESSOR SCHIFF'S EXPERIMENT,

As described by himself (Pflüger's 'Archiv,' xxx. p. 258).

[Translation.]

"Now if in a dog or cat which has been operated upon, *i.e.* by division of both posterior columns, lower down (*i.e.* opposite 4–5 cervical nerve), and comparatively recently, and which, in spite of the residual ataxy, can move about actively, the sigmoid gyrus and adjacent cortex be exposed, and stimulated with an induction current obtained by closing the circuit of a small Leclanché battery, *no trace of movement will be obtained in either the fore or the hinder limb*, not even a twitching, although the current be very strong. *Only the facial muscles* contract; and if the strength of the current be increased, *the neck muscles* also, when the centre for the head and neck is excited; this, too, setting the hind foot in action sometimes."[1]

MY REPETITION OF HIS EXPERIMENT.

'BRAIN,' *April* 1886, p. 60.—[Abstract of full notes.]

Experiment V.—Dog. Adult Mongrel terrier. *Sept.* 11*th*, 1885.—Left posterior column divided (*i.e.* opp. 4th cervical nerve). 12*th*.—Dog apparently well. 13*th*.—Marked paralysis of left forepaw. Lies habitually on left side. In attempting to walk, frequently falls to left side (hemiplegia). 14*th*.—During the previous night symptoms of spinal meningitis noticed.

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[1] The language in the two accounts is practically identical where placed in *italics*.

"2.20 P.M.—Both forelegs rigid, general rigidity of trunk, &c., with the other classical symptoms of meningitis. Dog anæsthetised with ether.

"2.45 P.M.—Both sigmoid gyri exposed within two minutes, and then stimulated as above described.

("I may here take this opportunity of stating, that in this series of experiments the apparatus of stimulation were an ordinary Du Bois Reymond induction coil, one Daniell cell [in one case, two cells were used] and usual platinum electrodes. The divisions indicating the distance of the secondary from the primary coil are given in centimetres; the strongest current, therefore, being at zero.)

Current.	Right Sig. Gyrus.	Left Sig. Gyrus.
10 cm.	Nil.	Nil.
8 cm.	Nil.	Nil.
6 cm.	Flickerings in left trapezius	Fibrillar twitchings in right trapezius and protrusion of limb with extension of forepaw.
5½ cm.	Ditto.	Ditto, stronger.
Interval, *i.e.* rest of five minutes.		
5½ cm.	Ditto. no spread.	Ditto, and spread to head, and right lower limb.

"'The difference was so extremely marked as not to need further investigation.'

"In fact a superficial examination would have led one to state that the right sigmoid gyrus, as far as the centres for *the limb- and trunk-muscles respectively were concerned, was inexcitable.* As a matter of fact, however, in this, as in all other experiments, the right cortex are perfectly excitable, as evidenced *by the active movements of the facial and cervical muscles* as low as the distribution of the fourth cervical nerve. I have already described the condition of the spinal cord in this case (see page 18), in which description it will be remembered that the degeneration of the motor tract was most marked. This case will thus serve as a typical illustration of the mode in which I believe Prof. Schiff's result is always produced. Of course it was impossible in this instance that the nerve energy should reach the left limb muscles."

I leave it to the judgment of my readers whether, with the exception of minute details, which are set forth at length in the account of my experiment, the result I obtained was not exactly the same[1] as Prof. Schiff's, according to his original account of it. I think it will be seen by the above quotation that the charge he so frequently brings against me of omitting to refer to his original articles on this subject is not only unfounded, but also actually recoils on himself.

As I have now shown that the result of our experiments are perfectly identical when the pyramidal tract was blocked, I shall now criticise in detail his experimental procedure, a criticism which I should not have thought of undertaking, had he not so frequently accused me of neglecting to acknowledge or quote (and indeed read!) the sources whence I obtained my ideas of his views and methods.

(1.) Division of the posterior columns of the cord.

Prof. Schiff says that he effects this not as I naturally concluded from his continually calling it "Trennung" and "Durchschneidung," by a simple cut, but by performing a resection of a certain length of the column. This he describes distinctly in two places, the most recent of which I give first because it now appears that he has abandoned it for the older method. The later method he gives, Pflüger's 'Archiv,' Band xxx, page 257, five lines from the top.[2]

"Mehreren Hunden und Katzen habe ich, in der obern Cervikalgegend, die beiden Hinterstränge des Rückenmarks in ihren ganzen Breite durchschnitten und dann die Schnittwunde in der Tiefenrichtung durchziehen mit der Pinzette vervollständigt"!

The older method he gives in the 'Comptes Rendus,' 1854, p. 926.[3]

"J'ai fait une incision qui divisait transversalement la plus grande partie des cordons postérieurs près du bout caudal de la plaie, puis saisissant, avec une pince la partie coupée, j'ai pu à l'aide d'une traction douce, mais continue, en haut et en avant, isoler non seulement la partie coupée, mais toute la circonférence des cordons postérieurs dans la longueur de plusieurs centimètres"!

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[1] Note particularly the sentences in *italics* (mine).—V. H.

[2] *Translation.*—"In several dogs and cats I have cut through both posterior columns in the upper part of the cervical region of the spinal cord, and then deepened the wound by dragging out portions with forceps"!

[3] "I made an incision which divided transversely the major part of the posterior columns near the caudal extremity of my wound, then seizing the cut portion with forceps, I contrived, by employing gentle but steady traction forwards and upwards, to isolate not only the part divided, but also the whole mass of the posterior columns longitudinally for a distance of several centimetres."!

We learn from Prof. Schiff's interesting "Reply," p. 299, that he has abandoned his cutting (plus dragging!) operation for the combined cutting and excavating method I have just quoted from 'Comptes Rendus.' I cannot admit that either is an exact method of producing experimental lesions in the spinal cord; in fact, I cannot conceive of any operative procedure more likely to be followed by traumatic complications. I cannot anywhere find in Prof. Schiff's writing a distinct proof by microscopical investigation of the harmlessness of this method; indeed, he only says in 1854 that naked-eye inspection of the cord, after hardening in alcohol, revealed no damage to the surrounding parts, and therefore further discussion of this method appears to me waste of time. Although Prof. Schiff appears to have read my paper, he asserts that I divide the column by means of a puncture only; whereas, had he quoted me correctly, he would have said a puncturing incision. However, since I publish microscopical appearances at the point of section of each of the cords I experimented upon, I need not dwell further on my method, since any one can form their own judgment on this matter by referring to my drawings.

Prof. Schiff makes the somewhat indefinite suggestion that I damaged the pyramidal tract by pressure during the operation. I have not the least idea how such a thing could occur; but if we imagine it to happen, I submit that his method is equally open to the same error. I think I had better close the consideration of this part of the matter by stating that I gather Prof. Schiff's observations to have reference to my belief that the degenerative block in the pyramidal tract is partly brought about by tension in the small wound cavity in the cord, together with, of course, as I stated in my former paper, interference with its blood supply, &c.

However this may be, it is obvious from my specimens that when the operation of division of the posterior column was accomplished neatly and without complication, that there was no block in the pyramidal tract, and that, as a necessary consequence, Prof. Schiff's result was not obtained. The reader will no doubt now fully understand why I quoted Schiff's own experiment on p. 5, showing that damage to the pyramidal tract produced the very phenomenon which, in the same paper, he attributes to the ascent of a hypothetical degenerative influence to motor cortex from the divided posterior column.

Since I am compelled to be a little discursive in order to refute Prof. Schiff's charge, I must now rebut his statement, that the block I observed in the pyramidal tract, in those experiments in which his result was obtained, was not a true block, he suggesting that I said it was a sclerosis. Having thus fathered upon my description a misleading name, he tells us, p. 302, that he has previously demonstrated that sclerosis in man does not affect the axis-cylinders. All this byplay of Prof. Schiff has nothing to do with the point under discussion, because in my paper, which it is clear he cannot have read, to say the least of it, carefully, I state in full detail that the block is brought about by degenerative changes in the axis-cylinders themselves.

Prof. Schiff repeatedly charges me in his "Reply" with ignoring, or being ignorant of, his original statements. I have already shown that he must himself have forgotten his own writings, and I must now add that he appears ignorant of, or to ignore, mine also.

(2.) Clinical methods of examining the disturbances of tactile sensibility after the division of the posterior column.

Prof. Schiff takes exception to the means I adopted for testing tactile sensibility in my experiments. As the first, namely the clip, is a method which he himself devised, and upon which stress is laid not only in Dr. Huggard's paper, but also in his original article, I cannot conceive why Prof. Schiff finds fault with me for adopting it. Further, the second, viz. gentle touching with a point, is that which is universally employed all over the world for the purpose; and as to the third, Prof. Schiff refers to it in a comprehensive sentence of intended depreciation, viz.—"So it is evident that the mere enumeration of the three methods applied by Prof. Horsley do not warrant the exactness of his result." It is obvious that I cannot discuss such criticism as this.

(3.) How Prof. Horsley's results were obtained.

Prof. Schiff concludes his review of the experimental methods I adopted by a paragraph headed as above. Some points in his conclusion I have already shown to erroneously arise from an imperfect reading of my paper. I have now to finish all I ever intend to say on this subject, by rebutting the few facts finally alluded to by Prof. Schiff.

In the first place, although he admits that my lesions were generally very perfect, he nevertheless attempts to diminish their scientific value by pointing out that in one or two instances there was a minute fragment of the column left undivided. The general frailty of Prof. Schiff's position is here demonstrated by his drawing attention to this fact, since, as will be seen by reference to my paper, his result was actually well marked in these very experiments, while with one exception it was absent in those experiments in which the column was absolutely divided. I note further an expression in connection with this point, which Prof. Schiff uses—an expression which may mean a good deal, viz. "this must be so even in the cases when an incomplete slit increases by parietal inflammation." If Prof. Schiff really means inflammation, it is perfectly clear to my mind, that in his experiments strict antiseptic precautions are not successfully carried out. As in all my own researches, I reject all experiments in which healing does not take place by so-called immediate union, I searched Prof. Schiff's original articles, to find whether he followed the same course. It has not been in my power to consult the Italian papers he refers to, but not in the 'Comptes Rendus,' nor in Pflüger's 'Archiv,' where his original and most important papers were first published, nor in his 'Lessons on the Physiology of the Nervous System,' have I been able to discover reference to this experimental detail, which, is in my mind, of absolutely primary importance in planning experiments of this kind. I have dwelt on this point not so much with the object of discussing its importance as of drawing attention again to the fact that, in those of my experiments, which I considered failures, owing to the breaking down of the antiseptic treatment of the wound, Prof. Schiff's result was easily obtained; whereas, in the instances in which immediate union took place, and in which there was, of course, no "parietal inflammation" of the wound-cavity in the spinal cord, his result was either absolutely wanting or very slightly marked.

It is obvious to my mind, as I pointed out in my first paper, that such a fact as this, taken in conjunction with the further fact of there being an anatomical block in the pyramidal tract, makes my former conclusion, which I have in this paper demonstated to be confirmed by one of Prof. Schiff's own experiments, perfectly certain; viz. that his experimental method does not prove that section of the posterior columns of the cord causes a loss of function in the opposite so-called motor cortex.

In the second place, the mode of excitation of the excitable area of the cortex is a point upon which Prof. Schiff holds very definite views, and these he has given us in clear language: thus, he says, "I have already spoken of the method of irritation. The use of the induction current has produced so many errors in the physiology of the brain, and more than elsewhere under the hand of English physiologists, that at length it is time to give up the use of the induction coil—to return to the battery with rheocord which allows us to vary as we need the strength, the increase, and duration of the current. Elsewhere, page 8, he says: "The intensity of the current is a cause of active derivations,[1] and so we are never sure to localise well an induction irritation in an organ the form of which is not linear." He further goes on to suggest that, owing to the escape of current, motor centres, which he imagines to exist at the base of the brain, but which he has never been able to find, are irritated thereby. It is difficult to understand why Prof. Schiff wrote the first sentence I have quoted on this subject, because, apart from its somewhat curt criticism of English neurological experimentalists, any one unacquainted with the facts might easily imagine that it was not possible to vary the strength, the increase, and the duration of an induction current, which of course is absurd. As to the old objection that errors arise from escape of current, I think really, after the many years that this has been before the scientific world, it is hardly worth while to take further notice of it than to re-quote the most obvious confirmation of the value of the method, a confirmation which receives, by the way, additional strength from its being endorsed by Prof. Schiff's own late assistant, Prof. Löwenthal, in the paper published by him and Prof. Herzen, in the 'Archives of Physiology.' This confirmation of the accuracy of the method is simply as follows. If on irritating some given spot of the excitable area the movement produced thereby be recorded, it will be found that shifting the electrodes only of 1 or 2 mm. from that spot, a different movement will be elicited. Finally, that if an excessively powerful current be employed, the result will be summation of these two movements. It is therefore quite beside the mark to suggest that the induction current is unable to differentiate the relations of the function of one spot in the brain as compared with that of another. Prof. Schiff tells us that he uses a constant current. This has long since been abandoned by English workers in this department of science, on account of its electrolytic action on the surface of the brain.

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[1] Professor Schiff here clearly means escape of current.

I cannot help feeling, after all, that the discussion of this well-worn subject is quite superfluous, for I have shown that I obtained Prof. Schiff's result just as distinctly as he did himself, so that the difference between the constant and induced currents has no bearing whatever on the present question. I, in fact, have only touched upon it because Prof. Schiff objected to my employment of the latter form of irritation in the repetition of his experiments.

In conclusion, I cannot help expressing a certain amount of disappointment that Prof. Schiff, in taking exception to my repetition of his experiments, should have omitted to furnish the judicial reader with the full details of his operative procedure, with drawings of microscopic sections of the cord operated upon, &c., for I can find nothing more definite even in his original paper than "Mehreren Hunden und Katzen," &c., giving scanty details of only two of these, and finally winding up with "Die Autopsie der beiden Wunden des cervical Markes ist noch nicht gemacht." I submit, with all due respect, that to draw conclusions from experimental work of this kind is likely to be prejudicial to the progress of the physiology of the nervous system ; and that I do not feel this opinion to be in any way modified by what Prof. Schiff says in his present "Reply," for he still does not furnish us with any of the details I have referred to ; while I think I have said enough to justify my previous criticism. The said criticism amounting to this, that Prof. Schiff's experiment of dividing the posterior column of the cord, and subsequently discovering that on the fifth day after he could elicit no movement of the limbs on the same side by stimulating the opposite motor area, does not (owing to numerous inherent fallacies) *prove* the posterior columns of the cord to have a direct relation to that motor cortex. Further, that his result is only to be obtained when (as he himself has unwittingly demonstrated) there is damage to the pyramidal tract.

PARALYSIS OF THE MOVEMENT OF CONVERGENCE OF THE EYES.

BY H. PARINAUD, M.D. (PARIS).

(*Translated from the French MS. by* HENRY JULER, F.R.C.S.)

THE paralyses of the muscles of the eye,[1] as of those of other parts of the body, arise either from central or peripheral causes. Until the last few years this fact appears to have been ignored, for the descriptions are devoted almost exclusively to the paralyses of the third pair of nerves, and the symptomatology is exactly deduced from the distribution of these nerves and from the action of each muscle. Several works, however, have been published on this question, notably those on conjugate paralysis, paralysis of the sixth pair, and Hutchinson's ophthalmoplegia.

I may here be permitted to remark that in several publications, of which the first appeared in 1877, I have endeavoured to establish the distinction between peripheral and central paralyses. Hitherto these subjects have not seriously occupied the attention of oculists, but at the present time, thanks to the efforts of Prof. Mauthner, this gap in the literature of ophthalmology is becoming filled up.

In my paper on "Paralysis of Associated Movements,"[2] I have insisted upon the necessity of searching for *clinical types* which shall enable us to understand the significance of those partial and complex forms which are most common amongst central paralysis.

Associated paralyses, *i.e.* those referring to similar binocular movements, are central paralyses *par excellence.* One of these is well known as conjugate paralysis of the sixth pair, thanks to the clinical observations of Prevost, Foville and Priestley Smith; to a previous case with autopsy by Féréol; to the experimental researches of Duval, Laborde and Graux; and to a memoir by Landouzy, who enlarges the subject by examining the relations existing between the hemispheres and the bulbo-pontal nuclei.

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[1] The cases on which this paper was written were observed, either in Dr. Parinaud's ophthalmic clinique or in the Salpêtrière, where the author fills the post of Ophthalmologist to the Neurological Department of Prof. Charcot.—ED.

[2] 'Archives de Neurologie,' March, 1883.

Conjugate paralysis from lesion of the nucleus of the sixth pair of nerves is not the only associated paralysis that has been observed. This is easily conceived, inasmuch as the other binocular movements in virtue of their peculiar anatomical disposition are equally associated.

Amongst other forms of associated paralysis that I have studied, I have signalised the *Paralysis of Convergence,* of which I desire to delineate the characteristic features.

It is hardly necessary to remark that this central paralysis of convergence is distinct from the insufficiency of the internal recti muscles, with which we are familiar in connection with myopia, with peculiar arrangement of the cranial bones, and from a congenital muscular insufficiency.

The deficient converging power observed in exophthalmic goitre[1] by Möbius has always seemed to me to be in relation with the ocular protrusion. In certain cases it is true this symptom may appear before the latter is very manifest. But, though the disease be primarily of nervous origin, the loss of convergence in Basedow's disease must be referred to a mechanical cause, as Möbius suggests, and must not be taken for a paralysis strictly so called.

I may call attention here to one form of insufficiency of the internal recti which is in some sense intermediate between insufficiency from ocular and that from cerebral causes. It may be called *Insufficiency from over-fatigue,* from excessive cerebral, as well as muscular, exhaustion.

In July last year three young girls were brought to me from the Ecole Normale Supérieure. They all presented symptoms of muscular asthenopia, and all the characters of pronounced insufficiency of the internal recti muscles. None of the patients were myopic. The visual trouble at first developed towards the end of the scholastic year, and after the most assiduous labour. The use of prisms afforded some relief and enabled them to continue their studies. After the rest of the vacation, these symptoms all disappeared, and the use of the spectacles was discontinued.

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[1] Centralblatt für Nervenheilkunde,' 1886.

There are two forms of paralysis of convergence arising from central causes. The one may be called *essential* and the other *combined;* combined, because it coincides with paralysis of elevation and depression.

A.—Essential Paralysis of Convergence.

The vision of a near object is accompanied by three distinct muscular acts; The *convergence* of the eyes towards the object looked at, the effort of *accommodation,* and the *contraction of the pupil.* These three acts are concerned in the typical forms of essential paralysis of convergence.

(*a.*) Paralysis of the movement of convergence is indicated by two principal symptoms: the objectively appreciable defect of convergence and a peculiar diplopia.

In order to recognise the defect of convergence, we proceed in the same manner as for insufficiency of the internal recti. If the patient be told to fix the finger as it rapidly approaches his face, the eyes remain immovable; if the movement be performed, it is incomplete, the patient cannot continue to look at the object with both eyes, but one of the eyes turns outwards and he squints. Lastly, in making him fix a near object alternately with either eye, the eye which has been momentarily excluded from vision executes a movement of readjustment, in order to fix the object. These are the objective symptoms of insufficiency of the internal recti.

On the other hand, in the lateral movements the internal recti contract normally. In a word, the innervation of the internal recti is only concerned in the movement of convergence; it remains normal for adduction in displacements where the visual axes are parallel. The *diplopia* appears in the median plane at a variable distance from the subject; *it is crossed, and there is moderate separation of the images which persist without notable modification in all directions of looking.* The separation is not sensibly increased when the candle is moved laterally, sometimes it diminishes, a character which distinguishes this diplopia from that of all ordinary paralyses of the internal recti. The images not unfrequently become fused at four or five metres from the patient, a distance at which but little convergence is exercised, but cases do occur in which the diplopia persists at all ranges; this may be explained either by the degree of paralysis, or by the individual differences in the static condition of the eyes. There are subjects in whom, during absolute repose, the globes have such a tendency to diverge, that the maintenance of parallelism necessitates the intervention of converging force.

If the patient does not complain of diplopia when a coloured glass is employed, a prism, placed base upwards, before one of the eyes will generally cause the double vision to appear, and so allow its characters to be studied.

(*b.*) Accommodation is involved in this form of paralysis in various ways. In certain cases there is absolute paralysis in both eyes; in others there is more or less notable reduction of its amplitude: whilst in a third class the accommodation is normal.

Accommodation remains defective in monocular as well as in binocular vision. It cannot be admitted that it is indirectly associated in virtue of the functional association of accommodation and convergence. This explanation, more or less applicable in certain cases where the paralysis is recent, does not account for those in which the paralysis of accommodation is absolute and may persist indefinitely.

The double paralysis of accommodation is not accompanied by mydriasis from paralysis of the sphincter, which distinguishes it from certain facts of double paralysis belonging to ophthalmoplegia interna and derived from another process.

We may further distinguish paralysis of accommodation connected with paralysis of convergence from paralysis arising from reflex causes, or from those occurring after diphtheria in which there is no paralysis of the iris. In one case of double paralysis of accommodation, after diphtheria, which I recently studied from this standpoint, convergence and pupillary reflex were normal.

(*c.*) If in this form of paralysis there be no paralytic mydriasis, the pupillary reflexes are nevertheless modified, and mostly in a very characteristic manner. *The reflex is abolished for convergence and retained for light*, thus constituting a modification exactly inverse to that pointed out by Argyll Robertson as occurring in tabes. The reflex is equally defective when the patient is made to fix a near object with one eye only. In certain cases the pupil, though not dilated, will not react to any excitation.

PARALYSIS OF DIVERGENCE.

The association of ocular movements for fixation at different distances can be modified in another manner. Eyes may converge for short distances, but yet be unable either to bring their visual axes back to the parallel directions, or even to fix an object at the distance of one or two metres. This I have called *paralysis of divergence.*

Paralysis of divergence can hardly be recognised except by the character of the diplopia. This, in the median plane, only appears at a certain distance, *it is homonymous, with slight separation of the images, and persists without notable modification in all directions of looking.*

The paralysis of divergence is not incompatible with defective convergence. In some cases the visual axes are found to cross at a certain distance, whilst the amplitude of convergence is nil. Beyond this point the diplopia is homonymous; within it the diplopia is crossed.

Amongst other symptoms accompanying paralysis of convergence must be mentioned the difficulty of estimating distances and vertigo. The first of these is sufficiently accounted for by the paralysis of convergence and accommodation. This symptom is most marked when the visual trouble is rapidly developed. The vertigo is partly explained by the false notion as to the position of the object, and by the diplopia, but this is not its sole cause. *The majority of patients in whom this paralysis exists suffer from vertigo independently of their ocular troubles.* In some there is vertigo with a sense of translation in space, similar to that observed in Ménière's disease. It is, moreover, remarkable that aural troubles are sometimes found to be co-existent.

The essential paralysis of convergence consists, then, in the lesion of that function in virtue of which the point of convergence of the visual axes is displaced in fixation at different distances, a function which may be disturbed in two ways, by lesion of the forces of convergence, and of the forces of divergence.

As to the seat of the lesion which concerns this function, we are not able actually to arrive at a precise determination. There are some reasons for believing it to be situated in the cerebellum.

I would mention in the first place the condition of vertigo which I have found to exist in several patients, and which is independent of visual troubles. It is not necessary to recapitulate the rôle which the cerebellum is supposed to play in cases of this kind, and in the vertigo with sensation of translation of the Ménière type. Recent works render almost certain the existence of a special nerve, called *nerve of space* (" nerf de l'espace "), connected with the auditory nerve, the lesion of which produces the vertigo of Ménière. But it is not only peripheral lesions of the nerve that give rise to this; those of the cerebellum, to which it leads, can produce exactly similar vertiginous symptoms to those of the aural disease.

We form our notions of space not only by the sense of hearing, but also by that of sight. It is, however, especially by the movements of convergence of the eyes that we obtain our notion of the third dimension to which that of space is essentially allied. It is probable that this notion of space has a functional centre, whether perceived by the eye or the ear. If, in reference to hearing, there are reasons for placing this in the cerebellum, these facts are in favour of a centre in the same situation for the co-ordination of the movements of convergence.

Lastly, I will mention that the experiments of Duval and Laborde have clearly established the influence of the cerebellum upon the co-ordination of the movements of the eyes; and, a fact of importance from our present standpoint, this influence appears to bear exclusively upon the relations of the ocular axes to one another. Whilst lesions of the bulb and of the pons constantly produce conjugate deviations of the eyes, those of the cerebellum, and particularly of the vermis inferior, produces what these experimenters call *dissociated deviations*, in which the axes of the two eyes, instead of being displaced parallel to each other, are modified in their relations, so as to produce strabismus.

At the moment of sending this memoir to the press, I observed with M. Charcot a new case of paralysis of convergence, which has more strongly fixed our attention upon the relation of this paralysis to the vertigo of Ménière's disease (Obs. IV.).

The patient, in whom the visual trouble declared itself eight months ago, in what he terms an attack of congestion, had suffered from vertigo with diminished acuteness of hearing for fifteen years. The attacks, according to MM. Charcot and Gellé, who examined the state of hearing, presented the true characters of aural vertigo, and possibly the supposed congestion which marked the outset of the diplopia was nothing more than violent vertigo.

In the presence of such a case, the question arises whether a lesion of the ear could not give rise to visual troubles by reflex influence. We must always remember that this paralysis may exist for months, years, or even indefinitely, which is hardly the case with reflex paralyses; and besides that, it may exist without these violent attacks of vertigo, and without lesion of the ear. I am more disposed to believe that the troubles of hearing and of vision arise from the same central lesion.

I am hardly competent to solve this difficult question. I will only mention that Jackson has indicated the existence of abnormal movements of the eyes during aural vertigo, and that he attributes to them a certain rôle in the mode or origin of the access.

M. Charcot, on the other hand, has observed in patients the existence of diplopia during the access. Lastly, in some cases we may see ordinary paralysis persisting in certain muscles. One patient at the Salpêtrière presented at the same time as the paralysis of convergence, an absolute paralysis of the left external rectus muscle.

I must add that many patients suffering from typical aural vertigo present no symptom of loss of convergence.

OBSERVATION I.—*Paralysis of Convergence and of Accommodation Abolition of Pupillary Reflex and of Convergence.*

M. Vern—, æt. 30, mechanic.

Power of convergence, nil.

On attempting to converge, the left eye deviates freely outwards. Adduction normal during lateral movements of the eyes.

Crossed diplopia in the median plane for all distances. Beyond 3 mètres, separation of images diminishes; it does not increase lateral directions of regard.

M = −1 D in each eye. Double paralysis of accommodation. With + 2·5 D. reads No. 1 at 30 centimetres.

Pupils equal, of normal dimensions. No reflex for convergence. Active to light. Nor does the pupil react when he fixes with a single eye.

No lesion of fundus.

Appreciates distances badly, a circumstance which he much laments, as he was formerly an excellent shot.

Visual trouble commenced with diplopia three weeks ago. Headache five years. Vertigo three months. Erythropsia.

OBSERVATION II.—*Paralysis of Convergence and of Accommodation. Absence of Pupillary Reflex.*

Madame Bodd—, æt. 52.

Power of convergence, nil.

Contraction of internal recti normal in the lateral directions of regard.

Crossed diplopia upon the median line for all distances. When the candle is displaced laterally, the separation of images instead of being increased is diminished. Diplopia persists in all directions of regard.

H. = + 1·5 D. Double paralysis of accommodation. With + 5·5 D. reads No. 1 at 30 centimetres.

Pupils equal, freely dilated.

Both reflexes abolished.

Fundi normal.

Vertigo frequently since the age of 2. Patient always feels as if she were intoxicated.

Persistent pains in the occipital and left temporal regions. Knee-jerk normal. No ataxy, no syphilis.

OBSERVATION III.—*Paralysis of Convergence and of Accommodation. Abolition of the Reflex of Convergence.*

Madame Mita—, æt. 54.

Fixation of a near object gives rise to no change in the pupils.

Patient does not complain of diplopia. This can be produced by the simultaneous use of a red glass and a prism base upwards. At 4 metres the images are upon the same vertical line. Nearer to the patient there is crossed diplopia; the separation is not increased by lateral displacements of the candle. Double paralysis of accommodation H = + 2 D. With + 6 D. pp. at 30 cm. reads No. 1.

Pupils equal, normal in size.

Reflex retained for light, abolished for convergence.

Onset undetermined, vertigo for a long time. When the patient goes out in the bright sunlight she is seized with vertigo and vomiting. Sense of numbness on the left side.

Observation IV.—*Paralysis of Convergence and of Accommodation. Abolition of Pupil-reflex in Convergence.*

Mr. Ball—, æt. 42.

Movement of convergence, nil. The lateral movements have their normal amplitude, but are accompanied by slight nystagmus. Crossed diplopia for all distances and in all directions of regard. No notable modifications of separation in the right and left directions of regard.

Pupil-reflex reserved for light, defective when convergence is attempted or when a near object is fixed with a single eye.

Paralysis of accommodation in each eye. $M = -1{\cdot}25$ D. $V = \frac{5}{10}$. With 3 D. pp. at 30 cm. reads No. 1 Snellen.

No lesion of fundus.

Onset of visual trouble in October 1885.

After an attack, without complete loss of consciousness, the patient suffered for fifteen days from visual trouble which prevented him counting his fingers; since he has improved, he has remarked that he sees double.

For fifteen years the patient has had vertigo with slight deafness, in which MM. Charcot and Gellé have recognised the characters of aural vertigo.

Observation V.—*Paralysis of Divergence.*

Madame Lep—, æt. 31.

Neither strabismus nor trouble in ocular movements are objectively appreciable. *Homonymous diplopia* becomes evident at 40 cm. from the patient and increased beyond that distance. The separation of the images remains moderate; instead of becoming increased when the candle is moved to the right or left, it tends to diminish. The diplopia persists in all directions of regard, in the upper as well as the lower half of the visual field. No inclination of images.

The pupils react to light and to convergence, but feebly.

Sensibility of retina normal. No ophthalmoscopic lesions.

Sudden onset of visual trouble on Jan. 1, 1884, without loss of consciousness, without other accident. No vertigo. Habitually suffers from frontal headache. No ataxy nor syphilis.

OBSERVATION VI.—*Paralysis of Convergence and Divergence.*

Madame Dup—, aged 62.

All other movements normal.

Crossed diplopia on the median plane, for a short distance.

At 1.50 metre the images become fused. At 3 metres diplopia becomes homonymous.

Diplopia persists in all directions, without any notable modification for the same distance.

Pupils normal in size, and reaction.

Sudden onset of visual trouble by diplopia without any other symptom. No vertigo.

B. COMBINED PARALYSIS OF CONVERGENCE.

In this form of paralysis, the defect of convergence is accompanied by paralysis of elevation, and of depression in both eyes, with retention of lateral movements.

This paralysis appears to be very rare in its typical and complete form. I have seen one case where the visual trouble, which had come on suddenly in a polyuric patient, was clearly characterised. The following is a résumé of the observation, reported more fully in my memoir on *Associated Paralyses.*

OBSERVATION VII.—*Paralysis of Convergence with Paralysis of Elevation and Depression in both Eyes. Retention of Lateral Movements.*

M. Sey—, æt. 67.

Movements of elevation and depression almost nil in both eyes.

Complete paralysis of convergence.

The internal recti contract normally for adduction in lateral movements.

The diplopia is crossed or homonymous, according to the distance of the candle. The image of the right eye always remains rather the lower.

The elevators of the lids are intact.

No paralysis of iris; there is, on the contrary, slight myosis with abolition of reflexes.

The amplitude of accommodation is almost nil, but, knowing the patient's age, one cannot ascertain whether this be paralysed.

The paralysis appeared suddenly on the night of Feb. 8, 1881. Since that date there has been a tendency to fall to the left. Polyuria recurring in crisis, with heaviness of the head, from which the patient suffered before the development of the ocular trouble. Pulse 52.

In an observation by Wernicke, quoted by Nothnagel, we find the same type of paralysis. The two eyes are unable to move either upwards or downwards, whilst lateral mobility to the right or left remains perfectly normal. But the condition of the convergence is not pointed out.

This form of paralysis may be incomplete. With paralysis of convergence we may have only paralysis of elevation (Observation V. in my memoir), or paralysis of depression (Observation by Priestley Smith[1]).

These symptoms correspond exactly to those of paralysis by lesion of the nuclei of the 3rd pair, as we take it to be along with integrity of the nuclei of the 6th pair, which are well known to innervate the external rectus of the same side and the internal rectus of the opposite side, but only for the lateral movements.

The extension of the paralysis to both eyes may be explained by the alteration of the two nuclei which are sufficiently near to each other to be simultaneously attacked by the same lesion. Moreover it is not impossible that the lesion of a single nucleus may produce bilateral results. This hypothe sishas already been advanced to explain certain cases of labio glosso-pharyngeal paralysis (oral communication by M. Charcot[2]).

Finally, it is not impossible that the lesion, instead of directly attacking the bulbo-pontal nuclei, is situated in a neighbouring centre, which acts immediately upon them, and it appears that this centre may be the tubercula quadrigemina. This appears to be the result of Wernicke's observation (paralysis of elevation in each eye, with conservation of lateral movements), where the autopsy proved a lesion of the *right corpora quadrigemina*, and of another by Hénoch[1] in which the paralysis of elevation in each eye was connected with a well-defined tubercular lesion of the *left posterior quadrigeminal body*.

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[1] 'Ophth. Hosp. Reports,' 1876.

[2] I do not know of any very conclusive observations on paralysis of the 3rd pair relating to one eye only in which *the movements of adduction of the internal rectus are conserved* in the lateral directions of looking. Therefore in considering typical nuclear paralysis we must suppose each nucleus to innervate one eye only. The following remarks bear upon a case in which this form of paralysis was well characterised, but the affection appeared to be congenital:

It has been long known that connections exist between the oculo-motor centres and the quadrigeminal bodies. In this direction we shall bear in mind the recent experiences of Darkchewitch,[2] who confirms and delineates the existence of these connections.

OBSERVATION VIII.—*Paralysis of the 3rd pair, with Conservation of the Movement of Adduction, probably Congenital.*

Marie Bauv—, æt. 12½.

Left Eye.—Movements normal in monocular and binocular vision.

Right Eye.—In monocular exploring, the movements of elevation and depression almost nil. Adduction and abduction normal. Action of inferior oblique appears to be completely defective. Action of superior oblique, deprived of its antagonist by the movements of torsion, is, on the contrary, exaggerated. If the patient attempt to look downwards and outwards the eye describes a downward movement, and further undergoes a very marked degree of torsion, similar to the hands of a watch.

In exploring binocular vision the same results are found, and further that the movement of convergence, which is normal in the left is absent in the right eye; it is true this eye is highly amblyopic, only counts fingers.

No lesion of the fundus.

The pupil is slightly dilated, but there is no paralysis of the sphincter. The reflex is absent for convergence, it is feeble for light. The elevator of the upper lid is not affected. The affection appears to be congenital; the child has always squinted freely; she had convulsions at the age of 26 months.

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[1] 'Berlin. Klin. Wochensch.,' 1864, No. 13.

[2] 'Neurolog. Centralblatt,' 1885.

NOTE ON TWO NEWLY DESCRIBED TRACTS IN THE SPINAL CORD.

BY CHARLES S. SHERRINGTON, M.B.

It is now twelve years since Woroschiloff published, among other experiments conducted under Ludwig's supervision, some which showed that in the lower dorsal region of the spinal cord of the rabbit, after both posterior columns and the posterior halves of both lateral columns, and the whole of the grey matter lying behind the central canal had been cut through, sensory impulses arising in the nerves of the hinder limbs still passed forward to affect motor mechanisms in the cervical spinal cord and brain. The tract or tracts thus transmitting sensory impulses in the anterior (ventral) half of the spinal cord remained an anatomical mystery until much more recently. The direct cerebellar tract, if that occupy in the rabbit a position corresponding to its position in man, it could not be, for, in that case, by Woroschiloff's experiment, it must have been completely cut through.

Passing over an observation of Gowers in 1877, which tended to extend Woroschiloff's result to injuries of the human cord, and, passing over scattered evidence by Westphal of the existence of tracts capable of independent degeneration in the ventral portion of the antero-lateral column, especially of the cervical region of the cord, Gowers' notice of an ascending antero-lateral tract in his 'Diseases of the Spinal Cord,' is the first anatomical confirmation of Woroschiloff's observation. Westphal undoubtedly figured at least once a degeneration of the tract elucidated by Gowers, but his material did not allow him to delimit, nor could he suggest the function of fibres degenerated. Kahler and Pick, Strümpell and Leyden, also figured portions of the same tract, but had no means of gauging its real extent or its significance.

In 1880, however, Gowers figured a preparation taken from the dorsal region of a spinal cord in a case in which the lower extremity of the cord had been crushed. The preparation was figured to show that besides the ascending degeneration in the posterior columns, an area of ascending degeneration existed in each lateral column just in front of the region of the crossed pyramidal tract. This ascending lateral degeneration gave in cross section an area shaped like an inverted comma, the tail of the comma occupying the periphery of the antero-lateral column in front of the direct cerebellar tract, the head occupying part of Flechsig's "mixed area of the lateral column." In this case the injury to the spinal cord had been too low down to implicate the direct cerebellar tracts. Recalling Woroschiloff's experimental and his own clinical observations, Gowers concluded that the tract in question subserved cutaneous sensory impulses.

A year later than the appearance of Gowers' work, Hadden showed before the Pathological Society of London specimens of a symmetrical degeneration in the anterior part of lateral columns. There was, however, no degeneration of the posterior columns, nor, unfortunately, any history attaching to the case.

In 1884, Déjérine published a paper, based upon two tabetic cases, in which he denied any true systemic character to the lesion occupying the periphery of the antero-lateral column. It is, he said, merely a chronic superficial myelitis secondary to a chronic pial meningitis, and propagated by continuity of tissue from the overlying pia mater to the underlying nervous structures. He termed it part of an annular "méningo-myélite corticale à marche subaiguë ou chronique suivant les cas." Against this view Strümpell had spoken, and Westphal has since urged the objection, that in two cases in which the peripheral degeneration had been found and figured by him, there was no thickening or other anatomical sign of inflammation of the pia mater, certainly not a trace in the region covering the degenerated area.

It is difficult to see how a meningitis could give rise beneath it to the symmetrical comma-shaped degeneration recorded by Gowers, for the head of the inverted comma lies removed from the periphery of the cord. And Hadden's case, if related to the others, exhibits the deeper portion of the tract suffering without participation by the peripheral. And yet stronger is the argument furnished by the observation of Bechterew, that a tract of fibres corresponding in position to a portion of the comma-shaped tract, develops apart from the rest of the lateral column.

Quite recently also further evidence in favour of the truly systemic nature of the lesion has been produced by Tooth. In a case of compression of the cord at the fifth dorsal vertebra, ascending degeneration had not only affected the posterior columns and the cerebellar tracts, but also areas situate at the periphery of the lateral columns near their junctions with the anterior. Still more lately has Westphal figured a border-degeneration (Rand-degeneration) in a case of diseased posterior columns reported at length in the 'Archiv für Psychiatrie'; and Strümpell in the same journal described and figured a very striking, although not uncomplicated example of it. The latter authority remarks, that there was no trace of concomitant meningitis; that the degeneration occupied the whole antero-lateral periphery from the cerebellar tract behind to the direct pyramidal at the lip of the anterior median fissure; and lastly, that we possess no hint as to the function of the fibres involved.

Not without reason, therefore, did Gowers publish, in the 'Neurologisches Centralblatt' of last March, a new note on his original case, adding that he had observed disease of the same tract in a couple of cases of tabes dorsalis.

To consider more fully the observation of Bechterew.

The original description by Türck, &c., of secondary degenerations of the pyramidal tracts, &c., acquired much greater weight when it became evident that the degeneration method and Flechsig's developmental method arrived at similar results. Flechsig, however, in the developing human cord, discovered no such tract as that found degenerated by Gowers. After distinguishing in the lateral column the crossed pyramidal and direct cerebellar tracts, he classed all the rest of the lateral column together as a "Vordere gemischte Zone." After distinguishing in the anterior column the direct pyramidal tract, he classed all the rest of it together as the Vorderstrang Grund-bündel. His work yields no evidence of the tract marked out by the comma-shaped border degeneration in the lateral and anterior columns. Recently, however, from material investigated in Flechsig's laboratory by Prof. Bechterew, the latter has been able to find in the lateral columns a tract of fibres developing apart from the rest of the mixed zone, and corresponding to a portion of the tract demonstrated by the ascending degeneration of Gowers; the portion thus confirmed is that bounding the direct cerebellar tract.

But the developmental method does in reality confirm, completely and in its entirety, the tract described by Gowers. The middle of the 8th month of intra-uterine life is the epoch at which to look in the human offspring for such evidence. The developing spinal cord then shows the tract precisely as delimited by Dr. Gowers. At that period there are three tracts, of which the nerve-fibres have not yet acquired their medullary sheaths; they are the pyramidal, crossed and direct; the tract of Lissauer adjoining the posterior corner, the comma-shaped antero-lateral tract. Of these three tracts Lissauer's is the first to become medullated; the pyramidal, crossed and uncrossed, the last. The comma-shaped antero-lateral has an intermediate date. But although the fibres of the comma-shaped tract may be spoken of as roughly all becoming medullate, at the same epoch, that is not strictly true. The more anterior (ventral) lying fibres acquire the medullary sheath earlier than do the more posterior (dorsal); also those nearest the crossed pyramidal tract acquire it earlier than do those at the margin of the lateral column. The fibres latest to develop are those at the margin of the lateral column, just in front of the direct cerebellar tract.

The tract is not marked off from the cerebellar by a sharp line, but groups of its fibres lie islanded within the more anterior (ventral) portion of the latter.

Probably Bechterew observed the tract at a period when its more anterior and internal portions were already medullate. Such a supposition accounts for his description of it, and the discrepancy between his observations and those of Gowers.

In figures 1, 2, and 3, of the plate, I give the appearance of the tract in the upper dorsal and cervical regions of the spinal cord at the 8th month of intra-uterine life. Fig. 4 shows it in the cervical region at a later stage, where its appearance agrees better with the description by Bechterew.

Stated briefly, the anatomy of the ascending antero-lateral tract is as follows. In position it stretches throughout the lumbar, dorsal, and cervical regions, occupying in cross-section a comma-shaped area, the head lying in the lateral columns touching the ventral edge of the pyramidal tract, the tail extending along the edge of the antero-lateral column from cerebellar tract behind, to anterior median fissure in front. At the top of the cervical region where the cerebellar tract comes to lie more anteriorly (ventrally) and the crossed pyramidal reaches the margin of the lateral column behind the cerebellar, the comma tract besides occupying the same area as it does at lower levels, stretches as a thin band between the cerebellar and pyramidal tracts.

The tract does not degenerate below a point of injury, but does degenerate above it. After injury about the level of the last dorsal nerve-root, its degeneration does not reach higher than the upper part of the cervical enlargement; a large proportion of the fibres composing the tract must therefore be confined to the spinal cord itself. Bechterew believes the tract at its upper extremity enters the nucleus lateralis of the medulla oblongata; it must be remembered, however, that he only observed the more posterior lying portion of the tract.

As to experimental evidence of the antero-lateral tract, Langley, in the April number of this Journal, drew attention to the probability that many of the fibres in the more ventral portion of the periphery of the lateral column, though they degenerate upward, are not fibres belonging to the direct cerebellar tract. In the mouse, after injury to the spinal cord at the level of the 4th lumbar nerve-roots, an ascending degeneration in the posterior columns and in the peripheral portion of the lateral column occurs, which, at a distance of nine nerve-roots above the injury to the cord, presents the localisation represented in fig. 5 of plate. There certain fibres are degenerating in Goll's columns, and certain fibres in the periphery of the lateral column, but not in that part of the periphery which, judging by analogy from the human and canine cord, belongs to the direct cerebellar tract. It is probable that the lesion was altogether too far down in the lumbar region to implicate the cerebellar tract, for I find nowhere good evidence of its degeneration. I imagine that the tracts degenerating at the level of the 7th dorsal nerve-roots (plate, fig. 5) are representative of the tracts that give in man the ascending degeneration in Goll's column, and the ascending antero-lateral degeneration of Gowers'.

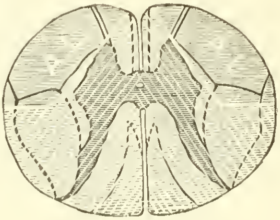
At the level of the 5th cervical nerve-roots, no degenerating fibres that I could detect were present in the cord of the mouse. The animal had been killed on the twentieth day after the operation upon the lumbar cord. On the supposition, generally admitted, that fibres secondarily degenerating in the spinal cord undergo that process almost, if not absolutely, simultaneously throughout their length, it follows from the above that the tract in question does not everywhere pass unbrokenly from cord to brain, although presumably those of its fibres whose distal organ is in the upper dorsal and cervical regions, do do so. The portion of the tract passing upward from the lumbar region does not appear to reach further than the lower cervical region. With regard to the function of the tract, the original experiment by Woroschiloff, the degeneration in an ascending direction, the clinical observations of Gowers, and the results obtained by Bechterew, all point to a sensory function.

I have repeated Woroschiloff's experiment three times—on dogs instead of on rabbits—and have supplemented the usual indices taken as evidences of sensation by observations on the blood pressure, after the plan adopted by Miescher. The three experiments yielded the same results. I quote the second of them.

June, 17, 1886.—Dog, ♂, weight, 14½ kilos.

10 A.M.—Chloroform, later chloroform and ether mixed, administered.

Spinal cord exposed through posterior arch of 9th dorsal vertebra; the posterior half of the spinal cord divided with a galvano cautery; a Beer's cataract knife then pushed forward (downward) through the thickness of the cord twice, each time with its straight edge in the median line, the first time the oblique edge toward the right, the second time oblique edge toward left. (In this way the only portions of the cord at the point of operation that remained unsevered were the two antero-lateral portions marked in the diagram by A L. A L.) Hæmorrhage stopped; wound covered by integuments; animal allowed to recover from anæsthetic.



Dorsal.

2 p.m.—Animal has recovered from anæsthetic, and has taken some food. Hind-limbs completely paralysed and flaccid; but pricking, pinching, and electric stimulation of the hind feet appear indubitably felt. The animal looks round to see what has touched him: hard pinching appears to cause discomfort, for the dog turns then quickly and angrily as if to bite.

2.15 p.m.—Chloroform again administered. Artificial respiration employed. Curare given by external jugular vein. The left carotid connected with a Ludwig's kymographion. The two sciatic nerves, right and left, prepared, and sheathed electrodes placed upon them. Time marker, seconds, and electric signal also registering upon drum of kymographion. Two pint Daniells in primary circuit of a Du Bois apparatus, for stimulation.

2.50 p.m.—Aortic blood-pressure as observed through left carotid is 11 mm. Hg.

2.55 p.m.—Stimulation for 3·5″ of right sciatic nerve, the secondary coil of the Du Bois (smaller) apparatus being at 10 cent. from the primary, causes blood-pressure to rise from 113 mm. Hg. to 127 mm. Hg. The curve of the rise as marked on the kymographion tracing shows two maxima, the earlier reaching 127 mm. Hg., the later—22″ later—reaching 124 mm. Hg.

2.58 p.m.—Stimulation, for 3″ of the left sciatic, with same strength of current, gives a similar rise of aortic blood pressure, showing again two maxima, an earlier of 129 mm. a later of 130 mm.

(These results were obtained several times over, and the experiment then concluded thus:)

3.40 p.m.—Abdominal aorta ligatured just below origin of lowest pair of lumbar arteries.

3.55 p.m.—Right sciatic stimulated with similar result to those recorded prior to ligature of aorta.

3.58 p.m.—Left sciatic gives similar result to right.

4.5 p.m.—Partial section of dorsal spinal cord turned into a complete division with the knife.

4.15 p.m.—Stimulation of sciatic nerves with secondary coil pushed over primary at full has no effect upon aortic blood pressure.

Thus sensory stimuli were able to initiate impulses that passed upward to the medulla through the portions A L of the cord at the ninth dorsal vertebra. Local vasomotor centres exist in the spinal cord, but that it was through these below the seat of operation that the rise of aortic blood-pressure observed in the experiment was brought about, is negatived by the persistence of the reflex rise in the line of the blood-pressure tracing after cutting out from the general circulation both hinder extremities and the pelvic viscera. The immediate effacement of the blood-pressure rise on turning the partial into a complete section of the spinal cord, again confirms the same conclusion. I may add that in one experiment the splanchnics on each side were divided, and that the reflex rise in blood-pressure persisted as before, although the general blood-pressure had of course become lower.

A tract of small fibres at the junction of the lateral and posterior columns has recently been described by Lissauer as containing all the small fibres entering the cord from the posterior roots of the spinal nerves. In view of Gaskell's late researches into the functions of the small fibres of the nerve roots, this description by Lissauer acquires the highest interest. The more so to the pathologist, that the tract is frequently sclerosed in tabes dorsalis. The independent existence of Lissauer's tract has also been this spring affirmed by Professor Bechterew, pursuing the developmental method of research. He includes it in the lateral column as a special constituent of that column.

From my own observations I find distinct developmental confirmation of Lissauer's tract, both in man and in the dog. About the middle of the eighth month of intra-uterine life in man, and in the newly-born puppy, a tract of fine fibres wedged in between the lateral and posterior columns begins to become medullate. By the ninth month these fibres are fully medullate. Until they are medullated, very few medullated fibres are to be found in the substantia gelatinosa of the posterior horn; a fact which confirms Lissauer's view of the identity in part of the two sets of fibres. The amount of substantia gelatinosa and the size of the area occupied between posterior and lateral columns by the small-fibre tract appear to me roughly proportional also, where one is large, so also is the other, and *vice versâ*. Bechterew also observed this relation. But to consider the tract a part of the lateral column as Bechterew does, involves a somewhat strained nomenclature. If the tract observed by Bechterew—he gives no figures of it—

be really the same as that described by Lissauer and observable in my specimens, it seems to me distinctly within the posterior column.[1] Indeed, according to my own preparations, it would seem to extend along the surface of the posterior external column for a varying but sometimes (lower cervical region) considerable distance toward the posterior median fissure. Lissauer's tract of fine fibres is easily seen in the crocodile's cord, and Dr. Gaskell has pointed out to me that in that animal the periphery of the whole of the posterior column is occupied by a zone of similar fine fibres. I find, as did Bechterew, that Lissauer's tract runs through the entire cord from the middle of the sacral region upwards. For the dorsal and cervical regions it is shown in figs. 1, 2, 3 and 4.

Finally, to return for a moment to the discrepancies in the hitherto published descriptions of the tract, called by Gowers the ascending antero-lateral. They contrast strongly with the unanimity of all descriptions of the pyramidal tract. Both pathological and developmental evidence point to the ascending antero-lateral tract being, as is in reality of course the crossed pyramidal, a congeries of smaller tracts, not all attaining their full development absolutely simultaneously, and, though close together, yet fairly independently localised. On the other hand, that all subserve analogous purposes and connect distant spinal segments in analogous fashion, one can hardly fail to believe.

REFERENCES.

GOWERS: 'Clinical Soc. Trans.,' 1878.
DÉJÉRINE: 'Archiv. de Phys. Norm. et Pathol.' ii. p. 454.
BASTIAN: 'Med. Chi. Trans.' 1867, p. 499.
WESTPHAL: 'Archiv f. Psychiatrie,' 1879, p. 413.
WESTPHAL: 'Archiv f. Psychiatrie,' 1880, p. 788.
KAHLER U. PICK: 'Archiv f. Psychiatrie,' 1880, p. 179.

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[1] In the lower lumbar region, and at the level of the lower extremity of the pyramidal decussation, the tract of Lissauer certainly does encroach upon the limits of the lateral column. That the size of Lissauer's tract in cross-section does bear direct proportion to the amount of substantia gelatinosa is only roughly true. At the top of the cervical region, for instance, the tract of Lissauer is as large as in the lumbar swelling. Again, the tract as one follows it from the spinal cord into the medulla oblongata, in which latter it at first bears the same relation to the substantia Rolandi as it does to the substantia gelatinosa in the former, becomes less and less distinct upward, although the substantia Rolandi progressively enlarges.

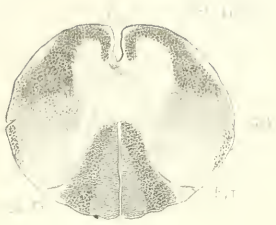

Fig 4

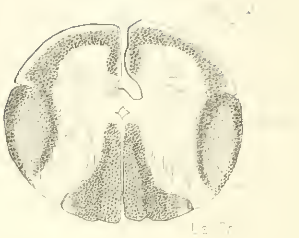

Fig 2

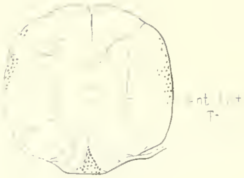

Fig 5

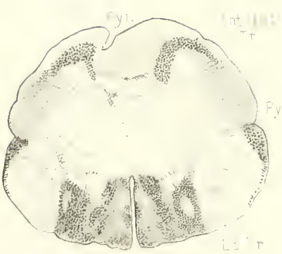

Fig 3

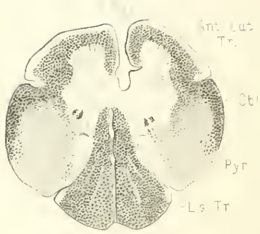

Fig 1

THE CAMBRIDGE SCIENTIFIC INSTRUMENT COMPANY

Strümpell: 'Archiv f. Psychiatrie,' 1880, p. 676.
Leyden: 'Zeitschrift f. Klin. Med.' 1880.
Bramwell: 'Diseases of Spinal Cord.'
Hadden: 'Path. Soc. Trans.' 1881.
Bechterew: 'Neurol. Centralbl.' 1885, p. 155.
Bechterew: 'Archiv f. Anat. u. Phys. His. u. Braun, Anatom. Abtheil.' 1886.
Gowers: 'Diseases of Spinal Cord,' 1880, 1st edition.
Gowers: 'Neurol. Centralbl.' May, 1886.
Gowers: 'Neurol. Centralbl.' April, 1886.
Howard Tooth: 'St. Bartholomew's Hosp. Rep.' v. xxi.
Singer: 'Sitz. ber. der Wien. Akad.' Bd. 83, p. 390.
Schultz: 'Archiv f. Psychiat.' Bd. xiv. 1883.
Löwenthal: 'Dissertat.' Geneva, 1885.
Langley: 'Brain,' April, 1885.
Gaskell: 'Journ. of Physiol.' vii. i.
Lissauer: 'Neurol. Centralbl.' 1885. No. ii.
Lissauer: 'Archiv f. Psychiat.' 1886.
Westphal: 'Archiv f. Psychiat.' 1886.
Strümfell: 'Archiv f. Psychiat.' 1886.

EXPLANATION OF PLATE.

Figs. 1, 2, and 3.—Cross sections of spinal cord at levels of 4th cervical nerve-root, of 2nd dorsal nerve-root, and of lower pyramidal decussation, respectively. From human fœtus at middle of eighth month. Coloured by Weigert's hæmatoxylin method, and magnified four diameters. Ad nat. del.

Fig. 4.—Similar to Fig. 1 above, but taken from the human fœtus of the ninth month.

The depth of shading represents the degree of development attained by the various tracts, the darker being the more completely grown. The only portions left white are the central grey substance of the cord and those tracts in the white matter whose fibres have not yet acquired medullary sheaths.

Fig. 5.—Spinal cord of mouse at level of 7th dorsal nerve, showing position of experimentally produced ascending degenerations.

Abbreviations used for the figures:—
Pyr. = pyramidal tract.
Cbll. = cerebellar tract.
Ant. Lat. Tr. = anterolateral tract.
L.'s Tr. = tract of Lissauer.

THE LATERAL RECESSES OF THE FOURTH VENTRICLE; THEIR RELATION TO CERTAIN CYSTS AND TUMOURS OF THE CEREBELLUM, AND TO OCCIPITAL MENINGOCELE.

BY J. BLAND SUTTON, F.R.C.S.,

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THE Pathological Society of London, on the evening of February 2nd, 1886, was concerned with the exhibition of a collection of intra-cranial tumours, which for richness and variety of material has probably never been equalled in the history of cerebral pathology. Among the specimens there shown, four especially arrested my attention. Two were exhibited as tumours in connection with the seventh nerves, the third and fourth as tumours of the cerebellum. The first two exhibited by Dr. Goodhart may be briefly described as follows: A tumour on each seventh nerve removed at a post-mortem examination in 1873. The case had been under the care of Dr. Wilks. The tumour on one side was as large as a pigeon's egg, whilst that on the other side was much smaller. The growths, which were of a fibro-sarcomatous nature, occurred in a woman, aged 26.

In the second case the tumour was of similar character, but unsymmetrical, and occupied the seventh nerve.

The third specimen was exhibited by Dr. Frederick Taylor as a fibro-sarcomatous tumour, growing out of one side of the flocculus of the cerebellum; it measured half-an-inch in length, was one-third of an inch in breadth, and a quarter of an inch in thickness, and gave rise to no demonstrable symptoms.

The fourth case, described by Dr. Beevor, was the brain of a man aged 40, with two tumours, one on either side of the medulla. The growth on the left side was of the size of a bantam's egg. It compressed the cerebellum and pons Varolii, and had by pressure destroyed the fifth, seventh eighth and ninth nerves. The tumour on the left side was of the size of a hazel-nut, and occupied the angle between the flocculus, pons and medulla. It has pressed upon the seventh and eighth nerves (Fig. 4).

These four specimens presented a set of common characters which set me thinking. They were all situated in the immediate neighbourhood of the flocculus, and of the seventh cranial nerve; they presented the same naked-eye characters, and agreed histologically.

In Dr. Goodhart's first specimen the disease was symmetrical, and this symmetry is by no means infrequent in this class of cases. In Dr. Beevor's case the tumours were symmetrical.

In addition to the above features the following fact is somewhat remarkable. ' Among all the neoplasms exhibited on that noteworthy occasion there were no other specimens in any other part of the brain which resembled these tumours, except one also exhibited by Dr. Goodhart as a fibrous growth of the left fifth nerve; but this I shall later endeavour to show had its origin from the same source as the preceding tumours.[1]

At that time the question of symmetry and asymmetry of disease was occupying my leisure time, and the nature of these growths seemed a profitable subject for investigation, especially as the normal anatomy of the particular portion of the brain, for which these tumours have a predilection, has long been a favourite study with me.

Turning our attention to the normal disposition of parts in the neighbourhood of the flocculus we shall find, as represented in Fig. 1, that the cavity of the fourth ventricle is extended laterally by means of two tubular prolongations, known as the lateral recesses, which pass between the superior and inferior peduncles of the cerebellum to open in the subarachnoid space on either side. These recesses are in close relation with the flocculus, and with the facial, auditory, glosso-pharyngeal and vagus nerves. The fibres of the auditory nerve after leaving the floor of the fourth ventricle, lie on the anterior wall of the recess. Prolongations of the choroid plexus pass through the recesses, and terminate in little villous masses at the base of the flocculus.

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[1] 'Many similar cases are recorded in periodical literature, and in the early volumes of the 'Transactions of the Pathological Society.'

These lateral recesses, and the important relations they establish between the subarachnoid cavity and the ventricular system of the brain, has not received the attention they deserve, either from the anatomist or pathologist. The credit of discovering this connection belongs to Key and Retzius, who have given a full description of the part in their monograph, 'Studien in der Anatomie des Nervensystem,' Stockholm, 1875. In the Erste Hälfte, Seite 111 und 115, "Ueber der offenen Zusammenhang der Hirnventrikel mit den Subarachnoidalräumen," the account is prefaced by a historical review, in which due credit is given to preceding anatomists, Bockdaleck, Reichert, and Luschka, regarding the recesses, but their real nature was detected by Key and Retzius.

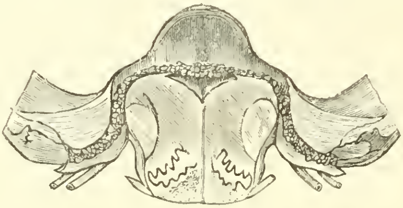

Fig. 1.—A transverse section of the fourth ventricle of the brain, showing the lateral recesses with their fringes of choroid plexus. The nerve filaments belong to the glosso-pharyngeal and vagus.

If the relations of the terminal extremities of the lateral recesses be inquired into, we shall find that they come into contact with that portion of the petrous bone indicated in the adult as the aqueductus vestibuli. A critical inquiry into this region discloses certain relations of no small degree of interest.

During the development of the internal ear, one of the most conspicuous features is a tubular prolongation, derived from, and remaining in communication with, the primary otic vesicle known as the recessus labyrinthi. This pierces the cranial wall, and becomes imbedded in the substance of the petrous portion of the temporal bone. Although very large in early fœtal life, it fails to grow at the same rate as the rest of the labyrinth; at birth it is represented as a narrow passage, terminated by a bulbous end, lined by epithelium, and known as the saccus endolymphaticus, and is lodged in the bony canal named aqueductus vestibuli.

Although in the later periods of fœtal life the saccus endolymphaticus is shut from the general cavity of the arachnoid by the dura mater, nevertheless in early embryonic life this duct establishes a communication between the labyrinth and the general arachnoid cavity. This communication, according to Professor Owen,[1] was known to Cotunnius.[2] These connections are of interest, for in the lateral recesses we have a means whereby the auditory labyrinth of both sides are brought into temporary relation through the intervention of the fourth ventricle. The saccus endolymphaticus in animals is apparently a remnant of an intricate system of ossicles and ducts discovered by Weber[3] in the loach, carp and sheat-fish (*Silurus glanis*), where, through the intervention of a sinus impar lodged in the basi-occipital, the swim-bladder is brought into communication with the interior of the labyrinth.

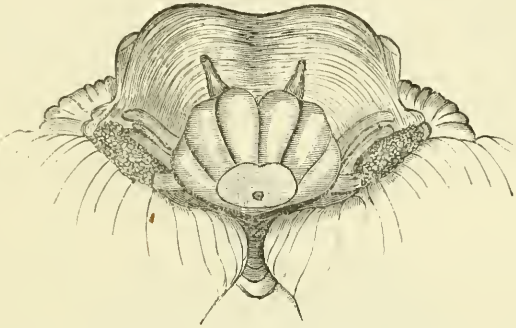

FIG. 2.—The pons and medulla represented somewhat diagrammatically, showing the tufts of choroid plexus emerging from the lateral recesses near the flocculus, between the seventh and eighth nerves anteriorly, and the glossopharyngeal posteriorly. (After Henle.)

Be this as it may, we have as matter of fact two tubular passages, leading from the cavity of the fourth ventricle to open into the general cavity of the arachnoid. These passages or ducts, like so many others in the body, are liable to become closed; in this case the obstruction is caused by a process of arachnoid occluding the outer end of the recess. The result of such occlusion is dilatation of the obstructed passage.

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[1] 'Anatomy and Physiology of Vertebrates,' vol. iii., p. 220, 1868.

[2] 'De Aquæductibus Auris humanæ internæ Anatomica Dissertatio:' 1774.

[3] 'De Aure et auditu Hominis et Animalium.' Lipsiæ, 1820, Part 1. 'De Aure Animalium Aquatilium.'

Besides stoppage of the recess this way, we must take into consideration the fringe of choroid plexus normally occupying its interior. It occasionally happens that the villi of the plexus enlarge, and thus constitute an obstruction, but more frequently they become the seat of a sand tumour, Psammoma. We thus get three distinct pathological conditions at the termination of the recesses. (1.) Cysts. (2.) Enlarged villi. (3.) Tumours.

(1.) It will be convenient to commence with the consideration of the cystic conditions. Virchow[1] figures a specimen bearing on the subject, and describes it as "a cystic hydrocele of the fourth ventricle with paralysis of the right facial nerve." Remnants of the choroid plexus were seen in the cyst. There was also hyperplasia of the pons Varolii and of the left hemisphere of the cerebellum. The case came from the Hospital of St. Hedwig.

The cyst was of the size of a cherry-stone and formed a projection on the surface of the brain, and clearly arose from the distension of that portion of the fourth ventricle known as the lateral recess. It pressed directly upon the facial nerve.

I have seen an exactly similar specimen in a brain which was dissected, whilst giving a demonstration on the anatomy of the brain to an ordinary dissecting-room class. On opening the fourth ventricle and endeavouring to expose the lateral recesses, the probe failed to make its appearance beside the flocculus, and on tracing the recess it was found to terminate in a *cul-de-sac* of the size of a nut. An example of the affection has been described by Recklinghausen[2] under the title of 'Doppelseitige Hydrocele der vierten Ventrikels.' The cysts were about the size of cherries. The author refers to Virchow's case quoted above. Between such simple cysts as these and the tumours represented in Fig. 4 is a variety of cyst with very thick walls. The following will serve as a typical example.

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[1] 'Pathologie des Tumeurs,' vol. i. p. 180.

[2] Virchow's 'Arch.,' Bd. xxx. s. 374.

The specimen is preserved in the museum of the Middlesex Hospital, and is of great interest, inasmuch as it was obtained by Sir Charles Bell, and the history of the patient, with a drawing of the parts, is given in his work on the 'Nervous System:'—

"The left Lobe of a Cerebellum and the Pons. Attached to the inferior surface of the left peduncle of the cerebellum close to its junction with the pons is a tumour the size of a pigeon's egg, consisting of a cyst with a distinct lining membrane, invested outside by a layer resembling convoluted brain substance. When recent, it was filled with a fluid the colour of urine. The fifth nerve, attenuated and flattened, appears to issue from the fundus of the tumour, and can be traced along its walls up to within half an inch of its origin. Both the portio dura and mollis of the seventh nerve are lost in the tumour from within a quarter of an inch of their origin as far as the meatus internus."

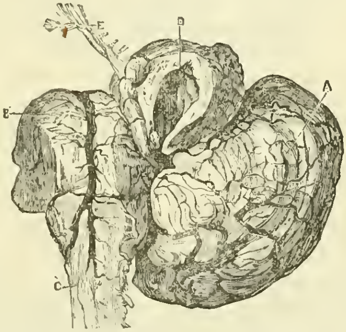

Fig. 3.—The under-surface of the cerebellum, showing a cystic tumour of the size of a pigeon's egg, involving the fifth, seventh and eighth nerves. (After Sir Charles Bell.)

(2.) From the cysts we must now pass on to the consideration of the tumours occupying this particular region of the brain. In doing so, one typical case will be described, and we shall then deal with the question of ætiology. As the type, I select that shown in the accompanying drawing, Fig. 4. It is the same case already referred to as being exhibited at the Pathological Society, by Dr. Beevor. Through his kindness and the courtesy of Dr. Ferrier I have been allowed to re-examine and figure the specimen.

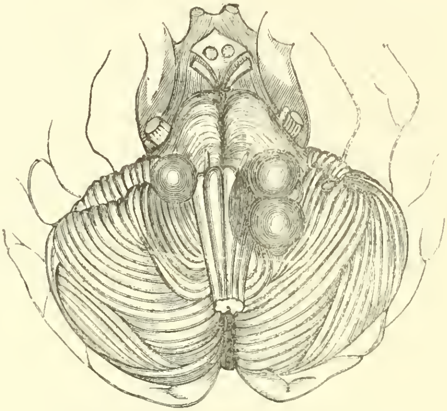

FIG. 4.—The under surface of the cerebellum. On the right side a large tumour is seen compressing the pons medulla, 5th, 7th, 8th, 9th, and 10th, nerves. On the left side a similar but much smaller growth is seen. (Diagrammatic.)

Examined microscopically, this neoplasm conformed in all respects to those singular growths so frequently found of small size, and so often symmetrical, in the choroid plexuses of the lateral ventricles, and known as psammomata. Their structure is often described as fibro-sarcomatous, because they consist of fibrous tissue sprinkled everywhere with round cells. But their most distinguishing feature is the deposit in them, often in concentric masses of gritty particles, sometimes in considerable quantity, imitating in this way the structure of the pineal body. Chemically, the earthy matter is made up of carbonate and phosphate of lime.

When we remember the extreme frequency of these tumours of small size, in connection with the choroid plexuses of the ventricles, not only in man but also of the lower animals, especially horses, their occasional occurrence in larger masses, and then recognised as psammomata, both in the brain and connected with the membranes of the spinal cord, and whenever they occur they are related to the arachnoid or its choroid plexuses, there need be little hesitation in ascribing the origin of the curious tumours described in the commencement of this paper to the cluster of villi situated in the angle formed by the flocculus, pons, and medulla. They agree with psammomata of the choroid plexus in the following points:—structural mimicry, symmetry of position, relation to arachnoid, and the age at which they occur, and the few clinical symptoms usually manifested by them.

(3.) It has long been a recognised fact, as Meckel pointed out in 1812, that cranial meningocele is by far the most frequent in the occipital region: in seventy-five cases tabulated by Laurence, fifty-three occupied this region. Another important circumstance connected with this deformity in the occipital region is the absence, or rudimentary condition, of the cerebellum.

All the cases of occipital meningocele or encephalocele which I have had opportunity of examining, serve to convince me that the cyst is in reality a distension of the cavity of the fourth ventricle. The accompanying drawing (Fig. 5) serves to illustrate this view of the matter.

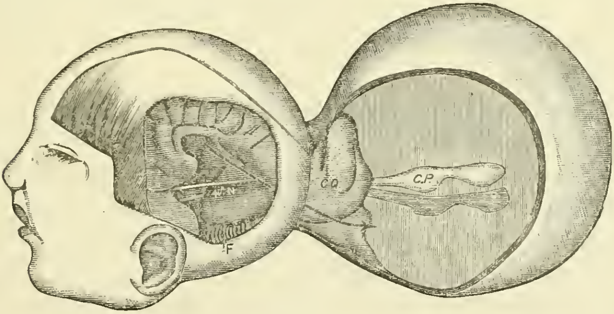

Fig. 5.—A ventriculo-meningocele. The tissue lining the cyst is continuous with the ependyma of the fourth ventricle. F. The flocculus (usually mistaken for a rudimentary cerebellum.) 4^{th}N. The fourth nerve. C.Q. The corpora quadrigemina. C.P. Choroid plexus projecting into the cyst. The arrow lies in the Sylvian aqueduct. (From 'Path. Soc. Trans.' vol. xxxiv.)

The details of the case may be briefly summarised as follows:—

The child survived its birth six weeks;[1] when it died, the cyst was as large as its head. In this specimen the lining membrane of the cavity is continuous with the walls of the fourth ventricle, and the cerebral matter projecting into it is probably the corpora quadrigemina; this view is strengthened by the fact that the fourth nerve is connected with it. The arrow indicates the *iter a tertio ad quartum ventriculum.* The fringes floating in the interior of the cyst represent the choroid plexus of the fourth ventricle. When I described this specimen originally, the cerebellum was said to be rudimentary, and this is the usual account given by those who have recorded cases of this kind. This is not absolutely correct, the cerebellum is absent, and the portion of brain attached to the medulla is, as a matter of fact, the flocculus, or sub-peduncular lobe.

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[1] 'Path. Soc. Trans.' vol. xxxiv. plate iii.

My attention was first directed to this matter by reading a careful and suggestive paper by Cleland[1] in which the following note occurs. "The flocculus, though always described with the cerebellum, is very well understood by those who have studied its development to be totally unconnected with that organ, being developed from a lateral outgrowth of the floor of the third cerebral vesicle, while the cerebellum is developed further forward from the foremost part of the roof of that vesicle."

This renders the question one capable of interpretation, for we have already seen that occlusion of one recess leads to cystic dilatation of the affected side. If both recesses become obstructed, it must necessarily lead to dilatation of the whole ventricular cavity, and occurring in early embryonic life, hinders the proper development of the cerebellum. Such a dilatation bears the same relation to a meningocele proper, as a syringo-myelocele bears to a spinal meningocele.

That this view offers a reasonable explanation of ventriculo-meningoceles in this situation, I venture to think few would doubt.

Virchow has described in his work on Tumours, in the part devoted to cysts, that occasionally one of the posterior cornua of the lateral ventricles becomes obstructed at the point where it joins the general ventricular cavity. As a consequence, the part behind dilates into a large cystic cavity.

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[1] "Spina Bifida, Encephalocele and Anencephalus." 'Journal of Anat. and Phys.' vol xvii. p. 257.

If this be true of the cornua of the lateral ventricles,[1] and cystic dilatation of the fourth ventricle may arise from obstruction to the lateral recesses, we can extend the notion further, and maintain that ventriculo-meningoceles, connected with the lateral and third cavities of the brain, may arise from obstruction to the aqueduct of Sylvius, occurring in early embryonic life, in the same way that it has been made to account for some forms of hydrocephalus.

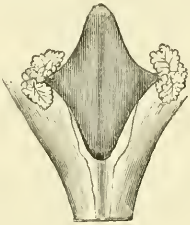

FIG. 6.—The fourth ventricle from a case of occipital ventriculo-meningocele. The cerebellum is absent. The foliated mass represents the flocculus or sub-peduncular lobe.

SUMMARY.

The contents of this paper may be stated in an abstract form as follows:—

1. Closure or absence of the lateral recesses of the fourth ventricle, in early embryonic life, leads to dilatation of the third cerebral vesicle, and give rises to a ventriculo-meningocele, projecting at the occiput.
2. If the lateral recesses become occluded after birth, a local cyst or cysts result.
3. The fringes of choroid plexus occupying the recesses may enlarge and form a villous tumour.
4. Such a tumour forms an excellent nidus for lime salts, and then become recognised as Psammomata.

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[1] Dilatation of the posterior cornua of the lateral ventricles of the brain are not uncommon.

ON CERTAIN QUESTIONS RELATING TO THE URINOLOGY OF THE INSANE.

BY ERNEST BIRT,

Senior Assistant Medical Officer. West Riding Asylum, Wakefield.

In a section of an important work,[1] published in 1884, Dr. W. Zuelzer treats of changes in the composition of the urine, associated with various conditions of altered metabolism in the nervous tissue.

He maintains that, from the nervous tissue, when in a state of lowered irritability, the delivery of material is augmented, and that it is lessened in conditions of exalted irritability. Further, that each of those series of conditions is, in respect to the tissue-change, differentiated by urinary qualities peculiar to it, and of such kind that, in depression conditions, (traumatic or pathological destructive brain-lesions; chloroform, ether, morphia, narcosis, &c.), the phosphoric and glycerinphosphoric acids[2] of the urine are increased; whereas, excitation conditions (as induced by strychnia, phosphorus, alcohol in small doses) are attended by a diminished amount of those products in the urine.

To illustrate the points claimed by Zuelzer, the following three analyses are selected from those contained in his work:—

Chloroform narcosis of 30 minutes' duration, for excision of adenoma. The urine voided immediately before the operation contained—[3]

N	P_2O_5	R
0·2648	0·076	28·7
	0·003	1.1

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[1] 'Untersuch. über die Semiologie des Harns,' S. 57, u.ff.

[2] Also the potash and lime salts; but these are beyond the scope of this paper so far as estimation of the basylous element is concerned.

[3] Throughout this paper the quantities found by analysis are expressed in grammes, or parts of a gramme. Where two series of figures are given under

An hour after the operation—

N	P_2O_5	R
0·38	0·173	45·5
	0·049	12·8

Morphia administered internally. The night urine of 21st–22nd July, contained—

4·96	0·89	17·9

0·02 g. morphia taken during evening of 22nd. Night urine of 22nd–23rd July contained—

4·85	1·218	25·1

Strychnia Experiment on Dog.

October 29*th*–30*th*.—Urine of—

2·2	0·358	16·2

October 30*th*.—0.002 g. strychnia subcutaneously.
During the spasms (a portion lost)—

0·15	0·011	7·3

October 30*th*–31*st*.—Evening and early morning,

1·74	0·164	9·4

October 31*st*.—Forenoon,

1·96	0·279	14·2

Mid-day,

3·43	0·58	16·9

Zuelzer further remarks that the alteration in the composition of the urine, associated with the first mentioned series of conditions, brings the relationship between the existing amounts of phosphoric pentoxide and nitrogen into more or less close accord with that which obtains in the nervous tissue itself, and with that present in the urine of animals (dogs) fed on brain substance.

According to his estimates, the normal gravimetric proportion of the P_2O_5 to the N, in the 24 hours' urine of an adult, is 18 or 20 to 100. In blood, the mean proportion is as 4 : 100; in muscle, 15 : 100; in brain and other nervous

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the heading P_2O_5, the first indicates the phosphoric acid in combination with alkalies and alkaline earths, the second, the glycerinphosphoric acid. The numbers under R denote the gravimetric ratio of the respective P_2O_5 to 100 N.

organs, which contain the greatest amount of lecithin, 45 : 100.

With regard to the glycerinphosphoric acid, concordant and definite estimates of the maximum quantity, present in the urine under normal circumstances, are wanting. In the 24 hours' urine of a healthy adult, Zuelzer found 0·06 gramme = 2·3 p.c. of the total P_2O_5, but he gives no numerical generalization upon the point.

Hoppe-Seyler[1] describes its occurrence in "very small quantity in normal urine."

Lépine and Eymonnet[2] place the normal amount at 0·25 to 100 N, or about 1 p.c. of the total P_2O_5.

These latter observers also have noted an increase in the renal excretion of the phosphorus compounds—particularly the glycerinphosphoric acid—as a result of gross cerebral lesions, epilepsy, and use of chloral or bromides. Thus in a case[3] of hæmorrhage into the external capsule and outer part of the lenticular nucleus, the urine excreted during the first six hours contained, per litre,

N	P_2O_5	R
2·5	0·54	21·6
0·0268	1·07	

"Forty-eight hours later, the proportion was normal."

With the view of ascertaining whether the above described urinary characteristics are commonly present in certain of the neuropathic, and psychopathic states met with in asylums, I undertook the subjoined analyses.[4] Especially, it seemed to

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[1] 'Handbuch der Phys. u. Path. Chem. Anal.' 1883, S. 116.

[2] 'Comptes Rendus des Séances de l'Acad. des Sciences,' t. xcviii., 1884, No. 4, p. 239

[3] Loc. cit.

[4] It may be well to designate the methods employed. In all cases the nitrogen has been determined in a Knop-Wagner's azotometer, by the process described in Zuelzer's 'Manual on Urinary Analysis,' using Dietrich's correction tables for absorption and weight. Zuelzer calculates that with this apparatus the hypobromite disengages the N to within 0·1, or at most 0·2 p.c. of the existing quantity, unless the urine contain much uric acid—2 p.c., or less, of the N may then remain in combination. With Hüfner's apparatus the deficiency is stated to be 7 p.c. The most favourable result obtained by use of the improved Simpson and O'Keefe's method left 0·09 p.c. combined (Cranstoun Charles).

The phosphoric acid united with alkaline earths has been estimated by precipitation with ammonia, and titration with standard uranic solution.

That in combination with alkalies has been similarly titrated, after precipitame that such investigations might throw some light upon the nervous metabolic processes underlying those transient hemiplegiæ, or monoplegiæ, with or without stupor, which frequently may occur during the course of general paralysis, without leaving any macroscopic clue to their origin.

Case I.—General paralysis, 2 years' duration. F. S., male, 43, habitually elated, boastful, the imaginary possessor of millions of pounds, three wives, &c. Was able to walk about until October 22, 1884; stupor, with rigidity of all the limbs, developed early on that morning.

The urine drawn at 1 p.m. (excretion of about 15 hours), contained—

N	P_2O_5	R	Remarks.
10·69	2·481	23·2	
	0·1805	1·6	Max. axillary temp. 39·1°.
From 1 p.m. to 10 p.m.			
4·231	1·2659	29·9	
	0·0188	0·4	
From 10 p.m. to 9.30 a.m., *October 23rd.*			
7·461	2·5344	33·9	38·4°.
	0		
9.30 to 6 p.m.			
5·337	1·6898	31·6	
	0		
6 p.m. to 11 a.m., *October 24th.*			
9·24	3·024	32·7	Emerging from stupor: recognises attendant and calls him by name. Head and eyes persistently turned to right. Left limbs flexed and rigid: right limbs fairly under control.
	0		

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tion by ammonio-magnesian solution—the known quantity of earthy phosphates being subtracted from the result.

For ascertaining the amount of glycerinphosphoric acid I have nearly always employed the indirect method—prolonged boiling of the urine with nitric acid, rendering alkaline with ammonia, precipitating by the magnesian solution, titration, and subtraction of the known amount of alkaline and earthy phosphates. The direct methods (Lépine, Hoppe-Seyler, Sotnischewsky) comparatively are complicated, and involve a great expenditure of time.

Repeatedly, when this organically combined acid has existed in traces too small to be measured with certainty by titration, the microscope has enabled me to detect it in the form of a few crystals of triple phosphate.

11 A.M. to 6 P.M.

N	P_2O_5	R	REMARKS.
7·357	1.2 0·12	16·3 1·6	39·4°.

3 A.M. to 9.30 A.M., *October 25th.*

6·15	0·929 0·103	15·1 1·6	38·7·

From 6 P.M. to 10 A.M , *October 26th.*

11·82	1·715 0	14·5	37·6°.

6 P.M. to 9.30 A.M., *October 27th*

10·097	1·1787 0	11·6	Left hemiplegia persists: less rigidity in palsied limbs. 36·5°.

11 A.M. to 6 P.M., *October 29th.*

	0·441 0·0784		37·8°.

October 30th.

10·706	0·9047 0	8·4	38·4°.

In this case, the large absolute quantity of the glycerinphosphoric acid on the first day, and the increasing elimination of the inorganically combined P_2O_5 during the period of stupor, are very prominent facts.

They certainly are independent of the food, for during those days the patient took only small quantities of milk, beef-tea, and egg—articles which he ingested freely when the stupor had passed off. Doubtless, the muscular tension and the pyrexia may have been the cause, to some extent, of the augmented excretion of the latter mentioned P_2O_5; but these conditions had not disappeared when the P_2O_5 elimination had permanently fallen much below the healthy standard.

The left limbs became flaccid, and remained paralysed till death on November 7, 1884.

There was no trace of focal lesion in the encephalon—beyond old adhesions of pia to cortex.

In the next two cases there exists a notable discharge of glycerinphosphoric acid in connection with the occurrence of paralytic motor phenomena, which contrasts strongly with the absence of that compound when the patients had regained, what may be considered, their normal state.

II.—General paralysis, 4 years' duration. A. F., female, 40, quiet, demented, cheerful and clean.

October 10*th*, 1885.—After brief prodromal emesis, right brachial monoplegia, with speechlessness, occurred to-day. No extra impairment of consciousness; understands what is said, and responds, otherwise than verbally. Paralysed limb rigidly semi-flexed at elbow.

The urine drawn 12 hours after onset of attack, contained[1]

N	P_2O_5	R	Remarks.
3·298	0·105	3·2	
	0.644	19·5	
	0·028	0·8	

October 14*th*.—12 hours ending at noon.

8·514	0·1345	1·5	Remains speechless. Limb less rigid: slight return of voluntary power.
	0·7768	9·1	
	0·013	0·1	

October 22*nd*.—24 hours ending at 8 A.M.

10·289	0·272	2·6	Four convulsive attacks yesterday: none since 2 gr. chloral were given. Convulsions were general: head and eyes to right. Patient can nearly articulate "Yes." Limb continues paralysed.
	1·559	15·1	
	0		

October 24*th*.—12 hours ending at 10 P.M.

4·43	0·0468	1·05	No more fits.
	0·4368	9·8	
	0·0052	0·1	

February 26, 1886.—1 A.M. to noon.

2·514	0·112	4·4	No fit since October. Completely recovered use in limb. Speech partly restored.
	0·272	10·8	
	0		

February 27*th*.—1 A.M. to noon.

2·77	0·108	3·8
	0·288	10·3
	0	

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[1] The first (topmost) group of figures shows the amount of P_2O_2 in combination with alkaline earths; the second, that united with alkalies; the third represents the glycerinphosphoric acid. This order is followed in all cases where the three series occur in this paper.

III.—General paralysis, 5 years' duration. E. C.,[1] female, 40. Very demented and dirty, fairly quiet, thoroughly self-satisfied.

November 27th, 1885.—Left brachial monoplegia came on in night. No stupor, convulsion, nor speech loss.

Urine passed at noon and 6 P.M. contained

N	P_2O_5	R	REMARKS.
1·055	0·0728	6·8	
	0·084	7·9	
	0·0056	0·5	

November 28th.—Night and forenoon excretion.

2·277	0·0748	3·2	Recovered free use in limb yesterday evening.
	0·2912	12·7	
	0·2329	10·2	

December 7th.—Night and forenoon (part lost).

2·967	0·246	8·2	No further attack.
	0·126	4·2	
	0		

February 8th, 1886.—Night and forenoon.

2·8256	0·27	9·55	Has remained in usual state.
	0·09	3·22	
	0		

February 9th.—Night and forenoon.

2·838	0·2275	8·01	
	0·0875	3·08	
	0		

February 10th.—Night and forenoon.

3·001	0·26	8·66	
	0·025	0·83	
	0		

So far as my researches extend, one does not constantly find an excretion of glycerinphosphoric acid following the convulsive attacks of general paralysis. Similarly in epilepsies, even when an enormous series of fits rapidly proceeds to a fatal issue.

IV. General paralysis, unknown duration. J. W., male, 38.

February 19th, 1886.—Severe general convulsions from noon until 1.10 P.M. Deep coma throughout. Ten minutes before convulsions ceased, 2·7 grammes of chloral per rectum.

Urine drawn at 10 P.M. = total excretion since convulsions began, contained

N	P_2O_5	R	Remarks.
2·393	0·14	5·8	
	0·042	1·7	
	0		

February 20th.—12 hours ending 10 A.M.

4·003	0·0725	1·8	No further convulsion. Recovered consciousness this morning.
	0·7395	18·4	
	0·0195	0·4	

V.—General paralysis, 2 years' duration. A. H., female, 35.

February 10th, 1885.—Two strong convulsive fits, from which quickly recovered.

Urine drawn 3 hours after the fits contained

0·919	0·045	4·8
	0·095	10·3
	0	

April 23rd, 1885.—Convulsed in forenoon. Urine drawn at 1.30 P.M.

0·891	0·066	7·4
	0·069	7·7
	0	

Next 2 hours

0·531	0·0		No fit since morning.
	0·1004	18·9	
	0		

VI.—Chronic mania, 21 years. Epileptiform seizures, 2 months. No permanent contracture or paralysis. M. A. P., female, 60.

January 29th.—Convulsed for 2½ hours—the spasms being strong and mainly limited to left side. 1·5 gramme chloral per rectum.

Urine drawn near end of attack contained

1·252	0·0786	6·2
	0·2824	22·5
	0	

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[1] This patient died in April, 1886. There was no local softening, nor could the naked eye detect any appearance to account for the monoplegia.

January 30*th*.—Urine of next 7 hours contained

N	P_2O_5	R	Remarks.
1·858	0·0982	5·2	No further convulsion.
	0·06	3·2	
	0·0109	0·5	

June 16*th*, 1885.—Fits recurred, with scarcely any intermission, for a period of 4 hours, ending at 3 A.M. 8 grammes of chloral, per rectum, during that time.

Urine passed at 2 P.M. (two previous evacuations lost) contained

	0·01		
	0·0225		
	0		

October 13*th*, 1885.—Strong convulsions from 5 P.M. to 7.15 P.M. 1·5 grains chloral at 7 P.M.

Urine from 6 P.M. till noon, *October* 14*th*, contained

2·533	0·0723	2·8	
	0·1034	4·08	
	0·0103	0·4	

VII.—Epilepsy, many years. A. M., female, 35. Much demented, but clean and useful, corpulent.

January 4*th*, 1886.—9 severe fits in 12 hours ending 5.30 A.M. No great prostration or prolonged stupor.

Urine of night and forenoon contained

3·18	0·288	9·05	Has daily taken 4 gr. alkaline bromide for more than a year.
	0·216	6·7	
	0		

January 5*th*.—Three fits from 1 A.M. to noon.

Urine of night and forenoon contained

3·22	0·0784	2·4	
	0·294	9·1	
	0		

VIII.—Epilepsy, since puberty. S. T. P., female, 22. Demented, irascible and pugnacious. Movements, spastic and sprawling. Great prostration after fits, feeble health, spare.

December 21*st*, 1885.—Eight fits yesterday; 2 in night. 3 grammes of chloral in night.

Urine of night and forenoon.

2·87	0·27	9·4	For many months has taken 3·5 grammes of potassium bromide each day.
	0·4212	14·6	
	0·0243	0·8	

December 22nd.—Such as could be collected during 24 hours, ending at 2 P.M.

N	P_2O_5	R	REMARKS.
3·53	0·3012	8·5	No more fits or chloral.
	0·3102	8·7	
	0·0094	0·2	

December 25th.—Three fits from 6.30 A.M. to 9.10 A.M. Urine drawn at 3.30 P.M. contained

1·709	0·2244	13·1	
	0·4048	23·6	
	0·0176	1·02	

January 3rd, 1886.—Urine from 4 A.M. to 10.30 A.M.

1·83	0·0925	5·05	No fit for 30 hours.
	0·3071	16·7	
	0·0074	0·4	

IX.—Epilepsy, since puberty. S. H. B., female, 36. Very demented, sluggish and awkward. Corpulent.

For five days the urine was collected in the night and forenoon.

January 23rd, 1886.

2·545	0·13	5·1	Two fits in forenoon. No medicine.
	0·403	15·8	
	0		

January 25th.

1·599	0·0825	5·1	Three fits in night.
	0·2425	15·1	
	0		

January 26th.

2·21	0·14	6·3	One fit in morning, one in afternoon.
	0·35	15·8	
	0		

January 27th.

2·027	0·185	9·1	No fit.
	0·2925	14·4	
	0		

January 28th.

3·211	0·165	5·1	No fit.
	0·78	24·2	
	0		

X.—Epilepsy, since puberty. M. P., female, 33. Quiet, industrious, imbecile. Fair general health.

January 16th.—Five severe fits in the afternoon; 6 during the preceding night; 3 in the following night.

Urine from 7.30 P.M. to noon, *January 17th*, contained

N	P_2O_5	R	REMARKS.
3·779	0.38	10·0	Takes 3 gr. of bromide daily.
	0·4192	11·0	
	0·0633	1·6	

January 29th.—Urine of similar period.

10·812	0·315	2·9	Two fits in night. None for 3 preceding nights.
	0·735	6·7	
	0		

January 30th.—Urine of similar period.

3·099	0·2142	6·9	No fit.
	0·4488	14·4	
	0·0102	0·3	

XI.—Epilepsy, many years. M. M., female, 30. Much demented, sluggish, feeble and obese. Average number of fits, 2 weekly.

Seventy-seven severe fits during the 48 hours ending at noon, January 31st, 1886. 9 grammes of chloral were administered in that time. Death occurred shortly before midnight, at the onset of a fit—convulsions having been absent for several hours previously;[1] but a fatal termination from pulmonary engorgement and œdema having appeared inevitable for many hours.

The urine drawn in the evening of January 30th, and on the morning of January 13st contained

2·142	0·085	3·96
	0·391	18·25
	0	

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[1] It is a noteworthy fact that, in this Asylum, during the past seven years the only cases in which chloral has failed to arrest convulsions before irremediable pulmonary hypostasis has developed, have been those in which the patient has not been taking a bromide, continuously, for a long period. The observation extends to 13 males and 17 females, who died in the epileptic status. Two of the male patients commenced the bromide just after coming to the asylum. Of them, one died within a week, the other within a fortnight. Two of the females terminated a long course of bromide a month, and five weeks, respectively, prior to the commencement of the fatal series. None of the other patients had undergone bromide treatment for many months, or years, before the last outbreak of fits occurred.

XII.—Epilepsy, 18 years. B. D., female, 21. Imbecile, robust general health.

January 29th, 1885.—59 strong fits last night—only 2 after a second 2 gramme dose of chloral per rectum.

The urine passed at bedtime of the 28th January, 1885, before the fits came on, contained

N	P_2O_5	R
2·208	0·2508	11·3
	0·3225	14·6
	0·0358	1 6

January 29th, 1885. Urine drawn at 11 A.M.

N	P_2O_5	R
1·765	0·0806	4·5
	0·018	1·01
	0	

Death took place a few hours later.

Professor Lépine and M. Jacquin have recorded[1] some interesting observations concerning the P_2O_5 excretion in certain cases of essential epilepsy and of epilepsy from alcoholism. They found the proportion of the P_2O_5 to the N much below normal on the days between the fits—once as low as 8·6 p.c. In those patients the proportion notably increased immediately after a fit, the rise being absolute, and chiefly due to an increase of the earthy phosphates. An augmentation of the earthy phosphates was also noted when the patients had merely experienced sensations of a fit being imminent, or had undergone an attack of vertigo.

Those special features cannot be definitely traced in the foregoing analyses—Cases IV. to XII. Frequently, indeed, the ratio of earthy to alkaline phosphates is there excessive; but that anomaly is almost constantly owing to the remarkably small absolute quantity of the latter. Thus, reference to IV., VI., VII., and XII. shows (not without exceptions) an extraordinary failure of the alkaline phosphates in the urine first collected after the nervous discharge. A like phenomenon is present in some of Lépine's analyses. It is necessary to observe, however, that Lépine investigated the whole 24 hours' urine, for several days in succession; consequently a full comparison of my analyses, cited above, with those of Lépine, cannot be made.

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[1] 'Revue Mensuelle de Méd. et de Chir.' tome iii. 1879, Nos. 9 et 12.

In the next two cases the 24 hours' urine was collected, save on certain days indicated. Here, also, are some manifest disproportions in the amount of the phosphates; universal in the first case, greatest during the acme of the convulsive period in the second, but also existing on days[1] when no objective evidence of a fit was present. (It is not known whether auræ occurred at those times.) This disparity is, again, mainly the result of diminished alkaline phosphates.

It should be noted that the second patient took less food after the fits commenced, and then suffered from malaise, lassitude, and headache, which prevented her from working.

XIII.—Epilepsy, many years. M. M., female, 41. Much demented, excitable, quarrelsome, and noisy. Feeble health; exceedingly macilent.

N	P_2O_5	R	REMARKS.
January 30–31*st.*			
3·894	0·315	8·08	One fit on Jan. 30.
	0·504	12·9	
	0·0157	0·4	
January 31*st–February* 1*st.*			
3·505	0·368	10·4	No fit.
	0·46	13·1	
	0·092	2·6	
February 3–4*th.*			
5·403	0·456	8·4	One fit on Feb. 3.
	0·57	10·5	
	0		
February 4–5*th* (some lost).			
4·584	0·264	5·7	No fit.
	0·286	6·2	
	0		
February 5–6*th.*			
4·0492	0·371	9·1	One fit on Feb. 5.
	0·2915	7·1	
	0		
February 6–7*th.*			
4·415	0·3444	7·8	No fit.
	0·3444	7·8	

XIV.—Epilepsy, since puberty. C. H., female, 35. Not much demented, never displays great excitement, is a useful ward cleaner. Fair general condition. Fits usually occur at the catamenial periods.

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[1] See Feb. 12, 13, 16, 18–20, and 27. On March 1 and 4 the ratio of earthy to alkaline phosphates is quite normal.

February 10–11*th.*

N	P_2O_5	R	REMARKS.
6·544	0·387	5·9	No fit for three weeks.
	1·29	19·7	
	0·043	0·6	

February 11–12*th.*—(Part lost).

4·925	0·36	7·3
	0·88	17·8
	0	

February 12–13*th.*

5·163	0·343	6·6
	0·686	13·2
	0	

February 13–14*th.*

6·7155	0·462	6·7
	1·188	17·6
	0	

February 14–15*th.*

8·451	0·532	6·2
	1·292	15·2
	0	

February 15–16*th.*

5·544	0·432	7·7
	0·864	15 5
	0	

February 16–17*th.*

5·7	0·324	5·6
	0·9	15·7
	0	

February 17–18*th.*

7·734	0·511	6·6
	1·095	14·1
	0·073	0·9

February 18–19*th.*

7·1	0·474	6·6
	0·948	13·3
	0	

N	P_2O_5	R	REMARKS.
February 19–20*th*.—(A portion lost.)			
5·465	0·441	8·06	
	0·686	12·5	
	0		
February 20–21*st*.			
7·23	0·346	4·7	
	1·314	18·1	
	0		
February 21–22*nd*.			
9·089	0·489	5·3	
	1·876	20·6	
February 22–23*rd*.			
5·836	0·504	8·6	
	1·152	19·7	
	0		
February 23–24*th*.			
5·608	0·395	7·01	
	1·187	21·1	
	0		
February 24–25*th*.			
8·88	0·408	4·5	
	1·292	14·5	
	0		
February 25–26*th*.			
9·018	0·528	5·8	
	1·254	13·9	
	0		
February 26–27*th*.			
8·06	0·504	6·2	No fit hitherto.
	1·008	12·5	
	0		
February 27–28*th*.			
11·127	0·648	5·8	One fit.
	1·8	16·1	
	0		
February 28*th*–*March* 1*st*.			
8·42	0.497	5·9	One fit.
	1·704	20·2	
	0		
March 1–2*nd*.—(Part lost.)			
4·897	0·424	8·6	Five fits.
	0·636	12.9	
	0·106	2·1	

March 2–3*rd.*—(Much lost.)

N	P_2O_5	R	REMARKS.
2·649	0·216	8·1	Seven fits.
	1.36	13.5	Glycosuria.
	0		

March 3–4*th.*—(Much lost.)

N	P_2O_5	R	REMARKS.
4·41	0·253	5·7	Three fits.
	0·897	20·3	Trace of sugar.
	0		

March 4–5*th.*—(Much lost.)

N	P_2O_5	R	REMARKS.
5·239	0·42	8·01	One fit.
	0·595	11·3	No sugar.
	0		

In a case of recurrent mania, some details of which are annexed, the alternating mental phases were to some extent characterised by change in the P_2O_5 renal excretion—the ratio of that to the N was constantly lower when excitement was absent. No organically combined P_2O_5 was found while the mental affection ran its usual course. As soon, however, as a depression condition became established (partial collapse from peritonitis, sopor induced by morphia), a large elimination of glycerinphosphoric acid occurred. Muscular activity and alimentation can each be excluded from the production of this phenomenon, because the former was reduced to the oppressed exercise of the circulatory and respiratory muscles, with infrequent emesis, and the latter was totally prevented by the inverted action of the stomach.

Analysis was made from all the urine that could be obtained in the 24 hours; on some days, little, or none, was lost. The earthy phosphates were not separated from the alkaline.

XV.—Recurrent mania, 3½ years. N. M., male, 44. When maniacal, incessantly restless, noisy, gay, and mischievous; almost sleepless for several days. During calm periods, industrious and orderly. Feeble health; heart fatty, spare condition.

September 30*th*, 1884.

N	P_2O_5	R	REMARKS.
7·057	1·937	27·4	Maniacal for three days.
	0		

N	P_2O_5	R	REMARKS.
October 2nd.			
8·5	1·7	20·0	
	0		
October 4th.			
6·518	1·4124	21·6	Mania subsiding.
	0		
October 8th.—10 A.M. to 5 P.M.			
1·951	0·472	24·1	Nearly free from excitement.
	0		
October 9th.—Night urine.			
1·756	0·315	17·9	
	0		
October 13th.			
7·446	1·585	21·2	Quite tranquil.
	0		
October 14th.			
3·481	0·5372	15·4	
	0		
October 15th.			
7·669	1·116	14·5	
	0		
October 16th.			
5·385	0·81	15·04	
	0		
October 17th.			
6·9	1·302	18·8	
	0		
October 21st.—6 A.M. to 11 A.M.			
3·966	0·8613	21·7	Relapse commenced yesterday. Peritonitis developed in night. 0·016 gr. morphia, subcutaneously, at 9.30 A.M.
	0·1953	4·9	

Comparison of the P_2O_5 excretion during the existence of severe melancholic emotional disorder with that obtaining when recovery had taken place, affords some interesting points in the two following cases. The period of mental disturbance is associated with a striking disproportion in the amount of the respective phosphates, which lessens during convalescence until the normal ratio is attained. This character is especially noticeable in the case of the male patient; in which, further, there is seen a significant discharge of glycerinphosphoric acid, at the time when the disturbance of cerebral function was greatest. Here, again, this disproportion is mainly the effect of a decline in the alkaline phosphates, though there is some decided absolute excess of the earthy phosphates on the first two days, in the case last mentioned.

Each patient received the ordinary diet, throughout. The man, at no time, displayed much energy; the woman was employed at light work in the laundry, when the acute distress subsided.

XVI.—Acute melancholia. J. C., male, 46. Admitted on Oct. 31, 1884, in a state of profound dejection, almost amounting to stupor; had inflicted a dangerous suicidal wound on front of neck a few weeks prior to admission. Stated that the impulse to destroy himself arose suddenly, and he made no effort to resist it. No delusion. Motionless and silent, until roused by other persons. In good bodily condition.

N	P_2O_5	R	REMARKS.
November 1–2*nd*, 1884.			
5·109	1·309	25·6	
	0·385	7·5	
	0·33	6·4	
November 2–3*rd*.			
8·211	1·197	14·5	
	1·05	12·7	
	0·03	0·3	
November 3–4*th*.			
5·363	0·7597	14·1	
	0·8104	15·1	
	0·0506	0·9	
November 4–5*th*.			
6·375	0·642	10·7	Less depressed.
	1·19	18·6	
	0		
November 5–6*th*.			
	0·534		
	1·036		
	0		
December 6–7*th*.			
9·164	0·8	8·7	Greatly improved: still morbidly quiet and inactive.
	1·0	10 9	
	0		

N	P_2O_5	R	Remarks.
December 8–9*th*.			
13·553	0·988	7·2	
	1·146	8·4	
	0		
January 2–3*rd*, 1885.			
8·8	0·48	5·4	Recovered.
	1·184	13·4	
	0		
January 4–5*th*.			
9·6	0·48	5·0	
	1·2	12·5	
	0		

XVII.—Acute melancholia. E. L., female, 23. Admitted Dec. 6, 1884, showed great melancholic agitation; imagined that some stuff had been given to her, which had dried up her inside; said she "felt right dried up, as though there were not a drop of anything in" her. Feeble and reduced.

December 10–11*th*, 1884.			
9·9	0·824	8·3	
	0·944	9·5	
	0		
December 12–13*th*.			
8·379	0·46	5·4	
	0·979	11·4	
	0		
December 13–14*th*.			
6·021	0·444	7·3	Much improved: calm.
	0·756	12·5	
	0		
January 19–20*th*, 1885.			
7·604	0·341	4·4	Quite well.
	0·874	11·4	
	0		
January 20–21*st*.			
8·77	0·403	4·5	
	1·2307	14·03	
	0		

Apparently, ether narcosis does not always liberate glycerin-phosphoric acid. In the first case, given below, total suppression of the alkaline phosphates was observed in the urine voided soon after the narcosis; four hours later, the excretion of those salts had reached an amount much exceeding that present in the urine before the ether was administered.

XVIII.—Ether narcosis, of about 40 minutes' duration, for removal of cystic tumour from breast. The patient, E. O., 71, was a feeble old dement; inactive, but a fair eater, and in good bodily condition.

Feb. 12*th*, 1885.—The urine, passed just before the narcosis was commenced, contained—

N	P_2O_5	R	REMARKS.
0·641	0·032	4·9	
	0·032	4·9	
	0		

Fifteen minutes after end of operation—

0·312	0·026	8·3	
	0·0		
	0·0		

Four hours later—

0·834	0·0475	5·6	Urine has a strong etherous odour.
	0·1128	13·5	
	0		

XIX.—Ether narcosis, of 20 minutes' duration, for amputation of digit. The patient was a fairly robust, middle-aged, woman. Two and a half hours after the operation, the urine had a powerful smell of ether, and contained—

0·0987
0·4794
0·0282

The number of the foregoing analyses, without doubt, is too small to permit of safe generalisation upon the special facts displayed in them. Their value also is diminished by the circumstance that the total renal excretion of several successive 24 hours' periods was not always obtained for investigation. The explanation of that defect lies in the difficulty of collecting the urine of insane persons. Even when the catheter is frequently and regularly employed, the patient occasionally will micturate in bed, and thus frustrate the attempt to secure that 24 hours' excretion. The more intelligent patients are prone to cause failure by simultaneously evacuating the bladder and rectum into the same vessel. To avoid cumbering literature with false data, which might insidiously originate from such causes of error, I frequently used the urine of somewhat indefinite periods.

Moreover, the question of the composition of the urine serving as an exponent of tissue change proceeding in the nervous system is beset with many conflicting facts and opinions. For instance, glycerinphosphoric acid is a decomposition product of lecithin—a substance which is abundant in nervous tissue, especially the grey matter. Were the occurrence of lecithin co-extensive with only that tissue, we should possess a sure index of some forms of nervous metabolism ; but lecithin also exists in blood, bile, pus, milk, and yolk of egg, hence the significance of an excess of glycerinphosphoric acid in the urine is much reduced. Indeed, Lépine[1] at first regarded the presence of that acid in the urine as a diagnostic sign of fatty degeneration of the liver. Since then, he has found it in some cases of grave anæmia, icterus, enteric fever, and acute pneumonia, besides the nervous cases previously mentioned. Zuelzer has met with it in diabetes mellitus and "paralytic dementia," in addition to the instances recorded at the commencement of this paper. One does not learn from the observers mentioned, whether the acid in question were daily excreted during the course of those diseases, or were only casually found.

Dr. Speck, in an elaborate article[2] upon the relations of mental activity to tissue change, gives the results of his investigations concerning the urinary excretion, and the consumption of oxygen, and the elimination of carbonic acid. He summarizes his conclusions as follows: "The final result of the experiments is this, that mental exertion produces no direct effect upon the general tissue change. The molecular processes in the brain, which constitute its basis, consequently, either are not oxidation processes, or they are so slight that they are inaccessible to our methods of research."

However true that may be with regard to physiological conditions, it surely is not legitimate to argue therefrom that pathological events in the brain cannot produce recognisable effects in the urine? Reading Homer, or criticising German and French scientific works (as in Speck's experiments), cannot be supposed to involve retrograde metabolic changes in the brain similar in nature, or equal in degree, to those which find expression in the phenomena of an epileptic fit, or of a non-hæmorrhagic apoplectic seizure?

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[1] See annotation in 'Lancet,' Sept. 23rd, 1882.

[2] 'Archiv für Experiment. Path. u. Pharmacol.,' Dec. 1881, Bd. xv. S. 81.

It would occupy too much space to discuss all Speck's objections[1] to the views enunciated by Zuelzer; one of them, however, is based upon the smallness of the amount of P_2O_5 in the brain, and requires some consideration. Following Bibra's data, Speck estimates that only about 4·1 grammes of P_2O_5 are contained in a human brain, and thus "an extraordinarily intense tissue change must be surmised if it should effect any considerable alteration in the normal daily excretion of about 3 grammes." But, apparently, Bibra and Speck have ignored the lecithin of the brain, which contains 8·798 p.c. P_2O_5 (Hoppe-Seyler), and which, according to Lépine's calculations,[2] raises the total P_2O_5 in a human brain, weighing 1350 grammes, to about 13·5 grammes.

That some of this should, by pathological action in the brain, attain excretion through the kidneys, and effect perceptible alteration in the composition of the urine, can scarcely be doubted. The following quotation from the valuable work by Dr. Cranstoun Charles[3] has an important bearing upon this point:—

"The embryonic brain is much richer in water and poorer in cholesterin, &c., than the developed brain. Further, we find that the lower a mammal stands in the animal scale the richer is its brain in water and the poorer in ethereal extract (chiefly cholesterin and *lecithin*), and accordingly the nearer does it approach to the embryonic brain, whilst the higher the animal's position the poorer is its brain in water, and the richer in the ethereal extract. Some of the constituents removed by the ether are probably compounds chiefly derived from retrograde metamorphosis, and as this is possibly most active in the highest brains, we can readily understand why the amount of ethereal extract increases in brains of high development. That it is indicative of activity may also be seen from the fact that the very active medulla oblongata and the continuously active spinal cord are poorer in water and very much richer in ethereal extractives than the brain substance itself. In the case of cholesterin, also, it has been shown (Flint) that the blood of the carotid artery is poorer in this body than that of the internal jugular vein."

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[1] They do not relate to the glycerinphosphoric acid—at any rate that body is not mentioned by Speck, and at the date of publication of Speck's article, Zuelzer had only recently made his discoveries respecting it.

[2] Loc. cit., 'Rev. Mens.'

[3] 'Physiological and Pathological Chemistry,' 1884.

Now, if these extractives be so intimately associated with the special manifestations of nervous functions, it is reasonable to suppose that their decomposition products must be eliminated when those functions are annulled by a descending metabolism. Cases I. to III. seem to indicate this.

There can be no doubt that all future analyses of the urine, made in connection with nerve states, should include determination of the glycerinphosphoric acid, the alkaline, and the earthy, phosphates—the amount of each being recorded separately.

Clinical Case.

ON SENSORY EPILEPSY. A CASE OF BASAL CEREBRAL TUMOUR, AFFECTING THE LEFT TEMPORO-SPHENOIDAL LOBE, AND GIVING RISE TO A PAROXYSMAL TASTE-SENSATION AND DREAMY STATE.

BY JAMES ANDERSON, M.D.,

Assistant Physician to the London Hospital.

G.B., AGED 23 years, a schoolmaster, was sent to me, on the 19th of August, 1885, by Mr. Waren Tay, whom he had consulted for loss of vision in his left eye. He told me he had been well till eighteen months previously, when he began to have attacks of a "peculiar sensation" passing down the right arm into the hand, and causing a sensation of swelling in the hand. The feeling, he said, then passed up the spine from the level of the shoulders to the head, spreading over the back part of the "brain" as a "cold sensation," not extending to the front. He frequently shivered when the fit was upon him; but the "peculiar sensation" was confined to the parts mentioned. At first the attacks were rare, once in six months; then they became more frequent, till he had one daily, and sometimes latterly as many as fifteen during the day. During the previous week he had had about four attacks daily. In answer to the question if he knew when the attack was coming on, he replied that he knew about a minute before by "a sensation in his mouth," "a rough bitter sensation," which lasted during the attack. He generally had an attack after any excitement. At first he always saw "a childish scene," meaning a scene of his childhood, the same with every attack, "a trivial scene, not worth remembering," but which always came up till six months before, when the "sensation," *i.e.* the peculiar sensation in the right arm, became more frequent. In this scene he seemed to hear the voices of a woman and children; but the scene and circumstances passed from him, he said, when the fit passed off. He showed some reluctance to talk about the scene; but when I pressed him in a subsequent interview to describe it to me, he said the woman was a neighbour, and that he was there to play with other children, and she was scolding them. The incident had actually occurred, apparently, but there was nothing to impress it upon him—"a trivial little scene, seems absurd that it should recur."

Although when he came to me the scene had ceased to recur, he still heard voices when he had the "peculiar sensation," not speaking to him but in conversation. There were never any chewing movements or smacking of the lips. This I ascertained from his relatives. The attacks, he said, would not have been noticed by those around, unless their attention had been called; they lasted "only a few minutes," recently they had been even shorter. At first he did not know what was going on around, but now he could tell. At a subsequent interview, and in *answer to a leading question*, he said that occasionally at the time of the "peculiar sensation" he had a sensation of smell which he could not define—not strong, "decidedly not pleasant," "did not last long, was not very bad, did not always come, and was not present at first, only for the past four months."

He was a tall, well-built man, somewhat inclined to stoutness, "had made flesh for two years." His features were somewhat thick and blurred, the mouth deviated slightly to the left, and opened more widely at the left when he showed his teeth. He did not fix with the left eye, which diverged slightly outward. He had lived chiefly in the neighbourhood of Boston, Lincolnshire, and for the previous eight months in Dorsetshire; had always been healthy, and had never had any venereal disease. He had been a teacher for ten years, and was evidently a thoroughly intelligent and thoroughly reliable man. His father and a brother died of consumption, the former at 42, the latter at 29 years of age. His mother, two brothers, and two sisters are alive and all fairly well. One sister died of empyema (?), and two others of typhoid fever. (*September* 1886.—Since writing the above I have examined one of the above sisters. She is suffering from rapidly advancing phthisis.)

His left eye was discovered to be defective in 1882, when he was examined by Dr. George Kirkwood, of Peterborough, before admission into St. Peter's College. He could read ordinary newspaper-type with it, however, and he suffered no inconvenience from it until the end of August 1884, a year before he came to me. At this time his eyes "broke down," his left eye pained him, and the sight of it grew steadily worse, till in October he consulted Dr. Kirkwood, who has kindly informed me that he found as follows:—"After the use of homatropine the vision of L. was $\frac{5}{200}$, with a hypermetropia of $\frac{1}{10}$, that of R. was $\frac{20}{200}$ with H. $\frac{1}{16}$. There was marked retinitis and irregularity of the disc in L. and congestion of the disc in R." Dr. Kirkwood applied empl. lyttæ to the temples, and gave him liq. hydrarg. perchlor. with iron. Three weeks later he prescribed spectacles, but with the correcting lenses, V. of L. was only $\frac{20}{200}$, V. of R. $\frac{20}{30}$ nearly. A few weeks afterwards my patient left St. Peter's College, and Dr. Kirkwood did not see him again.

Subsequent to this time the sight of his left eye continued to fail, and during the two months previous to his coming to me it had failed rapidly. In April 1885, *i.e.* four months before he came to me, the sight of the right eye failed rather suddenly in the course of a week; "spots in front of the music," so that he could not read quickly. *At no time* had there been headache, vomiting or convulsion, and only recently occasional giddiness.

Examination showed that his heart and lungs, his kidneys and other abdominal organs, were to all appearance healthy. The reflexes, superficial and deep, were normal, and the motor power of the limbs was unaffected. His speech was ready and articulation clear. He was right-handed and had always been so. Neither he nor his relatives could tell me anything as to the facial deformity, which resembled imperfect right facial paralysis, but was so slight, that when the face was at rest the only noticeable abnormality was the shallowness of the right naso-labial furrow. There was no apparent loss of sensation in face or limbs. This was carefully tested. Smell was normal with the right nostril, very defective or completely absent with the left. Common sensation good in both nostrils. Taste was apparently somewhat defective on both sides; but his answers were not decisive. The hearing distance for a watch was 5 feet with each ear.

As above noted, there was slight external strabismus of the left eye, noticed in 1882, and possibly present longer. Otherwise the movements of the globes were normal, and there was no ptosis. R. pupil 2½ mm. acted well to light and with accommodation.

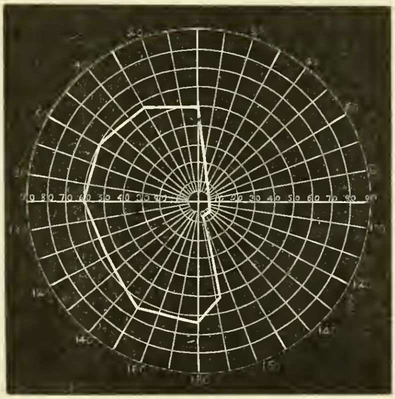

FIG. 1.—Right Visual Field of George B., Aug. 21st, 1885, for white object, 8mm. square.

L. pupil 3½ mm. acted well with accommodation, only consentaneously to light. V. of L. bare perception of light in temporal part of field, V. of R. $\frac{15}{20}$ partly and J. 1, also with + 2 D, the lens he was using. The R. visual field was of full extent on the nasal side, as shown in the accompanying chart, but crossed the vertical through the fixation point by only a narrow margin. The colour vision and nasal half of the colour fields of the R. eye were normal. *Ophth.—L.* Disc dull white, edge sharply defined. Veins of normal size, arteries very slightly diminished. No perivascular thickening or evidence of past retinitis. *R.* Outer half of disc atrophic, glistening white; inner half still pink, although not so pink as it should be. Vessels normal. No perivascular thickening or exudation.

On August 27th, Dr. Hughlings-Jackson kindly admitted him under his care at the London Hospital. Here the special senses of smell and taste were repeatedly examined, confirming the above results, the slight defect of taste being generally more marked on the left side. He took the bromide and iodide of sodium during his stay in the hospital, which he left on the 19th of October, 1885.

On October 23rd I again examined him, and made the following notes:—Mental condition much deteriorated, extreme hebetude, face expressionless, speech slow and monotonous, could not remember the word "hospital," and although wakening up occasionally into a state of much anxiety regarding his own and his mother's future, he yawned incessantly during the examination. His sister states that he rarely speaks, is generally in a somnolent condition, that he wanders, remembers nothing, and is always strange. He has had no headache, and vomited only once in hospital, from indigestion, he states. The divergence of the mouth to the left is now better marked. There is no definite paralysis or loss of general sensation. There is slight ptosis on both sides, especially the left, but both lids can be raised on request, and there is no ocular paralysis. V. of L. doubtful p l.; of R. $\frac{15}{70}$ and J. 16 with difficulty. R. visual field for a white object differed scarcely at all from that already recorded—temporal hemianopia. The fields for red and green were markedly and regularly contracted, especially the former. *Ophth.*—*L.* Paper-white atrophy of disc with small arteries and normal-sized veins. *R.* Inner part of disc still pink, but definitely paler than on last examination. Arteries and veins of fairly normal size. No other changes visible in fundi.

From this time till his death I visited him at his home. The mental hebetude gradually increased till his answers became incoherent, yet in flashes his mental state was perfectly clear. A brawny fulness came in the right side of his neck, which varied but did not disappear. He became considerably fatter and more somnolent. Vision got gradually worse, and the nasal half of the right disc got so pale that it could scarcely have been distinguished from perfect atrophy; would certainly have been called so by an observer seeing it for the first time. The arteries, however, remained of full size, in marked contrast to the diminished arteries of the left side.

On December 28th he had a "fit" on getting out of bed, became "stiff all over," and remained unconscious for half an hour. There were no chewing movements and no clonic convulsions. When he became conscious he vomited, and this was repeated frequently till I saw him next day. He apparently knew nothing of what had happened, and lay in a semi-comatose condition. There was no aphasia or evident paralysis. On examining the right disc I thought that its edge was slightly blurred as compared with its previous sharply-cut outline. On January 2nd there could be no doubt as to the blurring of the edge and fulness of the veins, although the centre of the disc was still white; the left disc remained in its condition of complete paper-white atrophy. On January 17th, after repeated vomiting and giddiness on the 14th, I found the right disc completely obscured by exudation, cloudy white in the centre with a considerable hæmorrhage at its upper edge.

From this time the vomiting recurred at intervals, with occasional attacks of flushing and perspiration followed by pallor. His limbs were stiff, the knee-jerks increased, ankle-clonus was present, and the superficial reflexes were abolished; he passed his evacuations under him. A few more hæmorrhages appeared on the right disc, the reflex from which was now as red as the rest of the fundus; and some glistening white spots appeared near the macula. There was no change on the left disc. Latterly he was probably quite blind; the movements of the globes were less free than previously; but there was no definite ocular paralysis.

On March 4th he passed rather suddenly into a state of complete coma. His left hand was occasionally raised to the left side of his head; the right lay almost perfectly powerless by his side; the knee-jerks were normal, and there was no ankle-clonus. He died on March 6th.

The ocular symptoms in this case pointed definitely to a basal tumour pressing on the left optic nerve in the first place, afterwards involving the left half of chiasma and left optic tract, and finally involving the remaining fibres of the right nerve. I believed also, from my acquaintance with Dr. Hughlings-Jackson's observations, that the paroxysmal taste-sensation and dreamy state, &c., justified the diagnosis that this tumour in some way, *i.e.* directly or through interference with vascular supply, involved the left temporo-sphenoidal lobe, inducing periodic discharges in the region of Ferrier's centre for taste (possibly also in his centre for smell), and in neighbouring highest centres. I use this last term in the sense in which it is used by Dr. Jackson. At this time I happened to mention the case to Mr. Nettleship, who referred me to a case published by him,[1] in which there were similar ocular symptoms. The similarity between the cases was, I found, surprisingly close, not only as to the ocular symptoms, but also in the occurrence of a paroxysmal sensation. Mr. Nettleship thus summarizes his case:—"Failure of left eye (to blindness) passing into atrophy of disc; later, paralysis of left third nerve, and loss of right half of right visual field with evidence of atrophy of disc; discharge of bloody mucus from left nostril, and late appearance of tumour behind left angle of jaw. Death seven years after onset of symptoms; large tumour compressing left optic nerve, chiasma, and tract, and left third nerve." Also it is noted that for several years before death he had been subject at irregular intervals to "fits," which consisted of a sudden feeling of suffocation in the nose and mouth, followed by pain in the front or back of the head, but no loss of consciousness. The similarity in symptoms between these cases was so close, that in writing Dr. Jackson I ventured to say that I believed the tumour would be found in the same position as Mr. Nettleship's, but involving a wider cortical area. The result, as will be seen, fully justified this diagnosis. Not only was the tumour in the same position, but, except in the matter of size, the description of the tumour in Mr. Nettleship's case might be used as the description in mine. Through Mr. Nettleship's kindness I have been able to examine the tumour in his case, and I believe it may be taken as an earlier stage in the development of the large tumour present in my case.

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[1] 'Ophth. Soc. Trans.,' vol. iv. p. 285.

Autopsy (March 7th, 1886).—Body extremely fat, two inches of adipose tissue on the abdomen; skull-cap thick, but readily separable from the dura mater. On the vertex the brain seemed perfectly normal; but on drawing it over to the right it was evident that there was a large tumour at the base. In removing the brain a large amount of ventricular and subarachnoid fluid escaped. Part of the tumour was adherent in the pituitary fossa, and this was removed subsequently. The adhesion of the tumour to the bone was not dense; little more than that between dura mater and skull. In the position of the pituitary fossa was a globular space floored by dense, convoluted, and somewhat roughened bone; its antero-posterior diameter was about 1¼ inches, its transverse about 2 inches, of which last half an inch lay to the right of the middle line, an inch and a half to the left. The part of the tumour remaining in the base of the skull extended slightly into the left orbit along with the structures in the left cavernous sinus and the left optic nerve, which were involved in it; the tumour-growth also passed through the left foramen ovale and foramen rotundum, which were about thrice their usual size. The right optic nerve and structures in the right cavernous sinus were pressed outward by the tumour, but were not directly involved in it.

The tumour is a large mass, consisting of a central soft greyish structure, occupying the position of the interpeduncular space, adherent in the pituitary fossa, and surrounded by masses of steel-blue blood cysts, as shown in the accompanying woodcut taken from a drawing, for which I am indebted to my friend Mr. Jonathan Hutchinson, Jun., and which was made from the brain in its recent state. The cysts extend only half an inch to the right of the middle line, but on the left they extend, large globular masses, backward and outward, undermining the posterior three-fourths of the temporo-sphenoidal lobe, which originally surrounded the cysts somewhat more than is indicated in the drawing. Anteriorly the tumour extends to the posterior orbital convolutions, and there is evidence of recent inflammation in the longitudinal fissure at this point; posteriorly it extends to the anterior border of the pons, and along its left margin, compressing considerably the left crus. The left olfactory nerve must have been early destroyed; but, owing to the softening and distortion of parts in this region, no part of the cerebral attachments of either nerve can be discovered. The right optic nerve, right half of chiasma and right optic tract lie clear of the tumour, although evidently much pressed by it. The left nerve and tract are so flattened out as to be almost indistinguishable on the anterior surface of the tumour imbedded among the cysts; the right third nerve lies on the under surface of the tumour, the left probably passes through it; but it is not visible, and was not seen during removal of the brain. The nerves behind this appear quite normal, including the left fourth nerve. The cerebral arteries, so far as they can be traced, are normal in appearance, the left carotid and its branches large, and imbedded among the cysts. The floor of the third

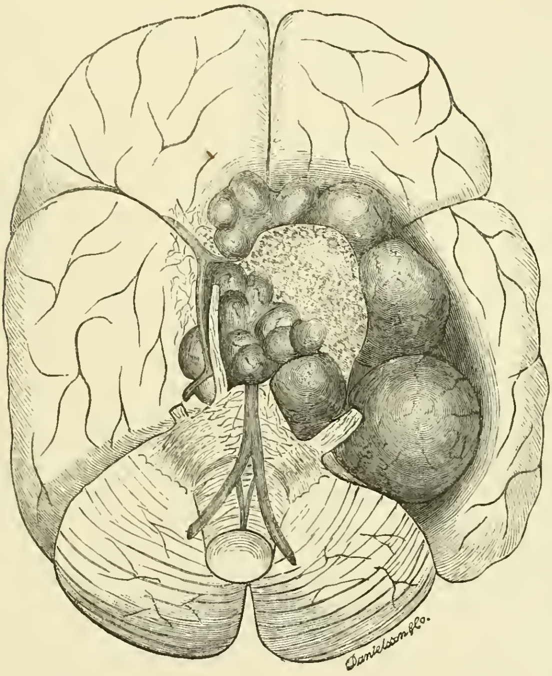

Fig. 2.—Base of Brain, George B., March 7th, 1886, after a drawing by Mr. J. Hutchinson, Jun.

ventricle is thinned to a mere membrane, and the ventricles were evidently greatly dilated; the cerebellum, pons, and medulla are apparently unaffected.

This case is interesting from various points of view especially taken in conjunction with Mr. Nettleship's. Let me emphasise a few of these points.

(1.) The tumour clearly arose from the pituitary body, and was in the first place at least of slow growth. The bone under and around the pituitary fossa was hollowed out, with the formation also of new bone in the neighbourhood. The foramina on the left side of the middle cranial fossa were enlarged, the nerves passing through them being surrounded by prolongations from the tumour, and apparently uninjured. In Mr. Nettleship's case death occurred seven years after the first symptoms. In mine it is impossible to decide as to the precise date of commencement, owing to the left eye being in all probability naturally defective from hypermetropia, but the duration of marked symptoms was less than four years. The central part of the tumour is a soft vascular mass, consisting of heterogeneous round and elongated nucleated cells without stroma. The surrounding cysts contain blood only, very indistinctly laminated, and have walls like those of a large vein. In Mr. Nettleship's case there is the same soft cellular growth in the centre, with the same blood cysts extending backward and to the left, the whole being probably in the recent condition about half the size of this tumour. The soft and cystic character of the tumour and its slow growth explain the escape in my case of the structures in the cavernous grooves which puzzled me before death. In Mr. Nettleship's case the left motor oculi was paralysed. As is generally the case in pituitary tumours, there was extreme development of subcutaneous fat and much mental hebetude, but there was at no time sugar in the urine.

(2.) The late occurrence of neuritis in the right eye was, I must admit, a surprise to me, notwithstanding that I was on the outlook for it. I had watched the disc pass into what would have been called complete atrophy by any one seeing it for the first time, and the eye was probably quite blind like the left. The right retinal vessels were of full or almost full size; but I expected to see them gradually diminish as on the left side. Nine weeks before death, however, there appeared in the right eye a neuro-retinitis, with considerable exudation and hæmorrhages. This occurrence coincided with the onset of vomiting and headache, both previously conspicuous by their absence. Had proof been wanted that this trio of symptoms depends in no way on the size of the cerebral tumour, and in very little degree at least on proximity of the tumour to the optic nerves, the present case would have amply sufficed.

Was the cause of these symptoms the slight recent inflammation of the meninges just in front of the tumour? Is local meningitis absolutely necessary to cause the trio of symptoms? I fear I cannot add much to what has been done in the way of answering this question. The meningitis in front of the tumour was certainly recent, and there was as certainly very little evidence of inflammation around the tumour at other points. Transverse sections of the optic nerves about a centimetre behind the orbits present, on microscopic examination, characteristic points of agreement and difference. The outer sheath is much alike in both, and, so far as I can see, in no way differs from normal. The inner sheath is more matted in the left, much more intimately blended with the periphery of the nerve, and sends into the nerve much larger trabeculæ. In neither right nor left inner sheath is there any noticeable increase of leucocytes; and I should not have recognised that of the right nerve from a normal sheath. The left nerve, notwithstanding its advanced atrophy, shows throughout fairly numerous axis-cylinders amid granular debris. The fasciculi are much shrunken, separated by thickened vascular trabeculæ, and traversed by a fine reticulum of stained fibres joining at intervals with the processes of branched cells lying among the axis-cylinders and debris—in fact, nervous tissue in a state of typical sclerosis. The right nerve shows but few axis-cylinders, in many parts merely a uniform granular debris, divided by thickened septa, which have no cell infiltration. The fascicular spaces thus marked out contain leucocytes, distinctly more than normal, varying in number in different parts of the section, but not uniformly more numerous at the periphery. At one side of the nerve (I am sorry I cannot say which side) there is an area in which the fasciculi contain branched cells, fine stained fibres passing among the granular material and axis-cylinders, precisely as in the left nerve. The neuritis, which appeared in the right eye nine weeks before death, was evidently of the usual type that occurs in cases of cerebral tumour, simultaneously with headache and vomiting. In ordinary circumstances it would have appeared also in the left eye; but the disc of this eye remained unchanged in its state of sharp-cut paper-white atrophy. Whether the immunity of the left nerve was due to the structural change in the nerve or its meninges, or to the practical disjunction of the nerve from the brain, this case cannot decide; possibly either would have been sufficient. In passing, I may say that the inflammation of the left fundus in 1884 was, to a thoroughly competent observer, compatible with simple hypermetropic change. It was doubtless, however, a neuro-retinitis of low grade, due to the retro-bulbar pressure, and leaving, as we have seen, little evidence of its presence except the slight opacity of the atrophic disc.

(3.) Lastly, the most important bearing of the case is on the doctrines as to the localisation of cerebral function. In this connection I wish to emphasise that there are two distinct clinical facts to be noted: viz. first, a paroxysmal sensation, and second, a dreamy state. The paroxysmal sensations in Mr. Nettleship's case and mine I consider to be precisely analogous to the motor manifestations of Jacksonian Epilepsy, and to be due to discharges in what Dr. Jackson has termed the middle sensory-centres, just as Jacksonian Epilepsy is due to discharges in the middle motor centres (the cortical part of which is known as Ferrier's motor area). The dreamy state I believe to be due to an extension of the discharge to highest cortical centres, which, as Dr. Jackson says, re-re-represent the body, in the complex combinations of which the reminiscence of my patient is an example. This dreamy state in such a case as this, is indistinguishable from epilepsy proper, or, rather, it is epilepsy proper, consisting, like it, of discharges in the " highest centres." It is analogous in mode of causation to the general convulsions and loss of consciousness, which closes a severe epileptiform seizure, but differs from it in the severity of the discharge, resembling in this latter point the cases of *petit mal*, where the discharge is sufficient only to produce a state resembling reverie.

To return to the paroxysmal taste-sensations which formed the starting-point of my patient's attacks. I have already stated that the tumour in this case arose from the pituitary body, and grew chiefly to the left and backward. I judge of this not only from the tumour itself, but also from Mr. Nettleship's case, which I consider shows an earlier stage in the growth of this tumour. From the position and relations of the pituitary body, it is clear that the first part of the cortex involved by the tumour would be the anterior part of the inner border of the temporo-sphenoidal lobe—in other words, the area in which Ferrier has had grounds for locating the centres of taste and smell, at the tip of the gyrus uncinatus. Producing by pressure an instability in the grey matter of this area, I believe that the tumour was the cause of the paroxysmal taste-sensations which commenced the attack, and that from this point the discharge spread to neighbouring centres.

Dr. Hughlings-Jackson has observed and noted, although not published, numerous cases of paroxysmal sensation, combined with dreamy state; and it was owing to my acquaintance with his views that I was led to investigate the present case in detail. McLane Hamilton has recorded a case of paroxysmal sensation of smell, in which chronic meningeal inflammation over the right uncinate gyrus and sclerosis of subjacent tissue was found *post mortem.* Such cases as these go far to substantiate clinically the accuracy of Ferrier's observation. They are probably more numerous than is generally supposed; this owing partly to the fault of the patient, partly of the physician. The patient may be stupid or careless in observation, and in the expression of what he has observed; or, on the other hand, like my patient, he may be unwilling to speak of it, as being "silly." I need hardly say that in all cases, but more specially with stupid patients, where the temptation is greatest, one must avoid putting leading questions. One such question may destroy altogether the value of subsequent examination. On the side of the physician, every medical man ought nowadays to be aware that the occurrence of these paroxysmal sensations or of the dreamy state is, as Dr. Jackson has shown, an important symptom, foreshadowing grave cerebral disease, as the first signs of tubercular change at the apex of a lung portend coming phthisis. In both cases the disease may be curable; in both it may be beyond cure. In either case it is our duty to know and recognise it from the first, and to guide our patient accordingly.

Critical Digests.

ON THE RECENT ADVANCES IN OUR KNOWLEDGE OF TABES DORSALIS.

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LITERATURE.

(1) Eulenburg's 'Realencyclopädie,' 2nd ed., 1885.

(2) "Zur Lehre von der spinalen Ataxie."—'Neurologisches Centralblatt,' 1885, No. 2.

(3) "The Relations between the Symptoms and Cord-lesions of Posterior Spinal Sclerosis."—'The Alienist and Neurologist,' vol. vi., No. 3.

(4) "Ueber die Bestandtheile der Hinterstränge des Rückenmarks auf Grund der Untersuchung ihrer Entwickelung."—'Neurologisches Centralblatt,' No. 2, 1885, p. 31.

(5) "Zur pathologischen Anatomie der Tabes dorsalis."—'Neurologisches Centralblatt,' No. 3, 1885.

(6) "Beitrag zur pathologischen Anatomie der Tabes dorsalis und zum Faserverlauf im menschlichen Rückenmark."—'Neurolog. Centralblatt,' 1883, p. 245.

(7) "Ueber einen eigenthümlichen Symptomcomplex bei Erkrankung der Hinterstränge des Rückenmarks."—'Archiv fur Psych., etc.,' Bd. xvi., 1885. Abstract in 'Neurol. Central.,' 1 Jan. 1886.

(8) 'Journal of Nervous and Mental Disease,' April, 1885.

(9) 'Ann. Méd. Psych.,' 7 Série, t. ii., Sept. 1885.

(10) 'Path. Trans.' 1885.

(11) "On the Combination of Lateral and Posterior Sclerosis of the Spinal Cord."—'BRAIN,' April, 1885, p. 110.

(12) 'Trans. Int. Med. Congress,' 1881.

(13) 'Neurol. Central.,' 1885, p. 241.

(14) 'Arch. de physiologie normale et pathologique,' 1884, No. 8, pp. 454–486. Abst. in 'Neurol. Central.,' 1885, p. 39: "Du rôle joué par la méningite spinale postérieure des tabétiques dans la pathogénie des scléroses combinées."

15) "Observations pour servir à l'histoire de l'Arachnitis et de la Leptoméningite spinale chronique."—'Sclérose médullaire secondaire. Gaz. méd. de Paris,' 1885, No. 324. Abst. in 'Neurolog. Central.,' 1885, p. 178.

(16) 'BRAIN,' vol. vi., p. 461.

(17) "Contribution à l'étude des lésions scléreuses des vaisseaux spinaux," etc.—'Rev. de Méd.,' 1885, Jan., p. 1. Abst. in 'Neurol. Central.,' 1885, p. 227.

(18) 'Rev. de Méd.,' July, 1885.
(19) "Ataxie locomotrice et lésions cardiaques."—'Montpellier Médical,' June, 1880.
(20) "Ueber die Coïncidenz von Tabes dorsalis und Insufficiens der Aorten Klappen."—'Berlinen Klin. Wochenschr.,' 7 July, 1879.
(21) "Ataxie locomotrice."—'Rev. de Méd.,' No. 2, 1886, p. 170.
(22) "Can Locomotor Ataxia be Cured?"—'Journal of Nervous Diseases,' April, 1885.
(23) "Ein Fall von Ataxie nach Diphtherie."—'Berl. Klin. Woch.,' 15 Dec. 1884.
(24) "Locomotor Ataxy without Disease of the Posterior Columns."—'Clin. Soc. Trans.,' vol. xviii., p. 168.
(25) 'Arch. de Méd.,' Oct. 1884. Abst. in 'BRAIN,' Jan. 1886.
(26) 'Encéphale,' Nov., 1884. Abst. in 'BRAIN,' Jan. 1886.
(27) "Sclérose primitive des cordons de Goll."—'Prog. Méd.,' 1884, p. 1008.
(28) "Recherches sur les causes de l'Ataxie locomotrice progressive." — 'Le Progrès Médical,' August 29, 1885.
(29) 'Berliner Klinische Wochenschrift,' No. 32, 1883.
(30) "Beiträge zur Aetiologie und Therapie der Tabes dorsalis, namentlich über deren Beziehungen zur Syphilis."—'Virch. Arch.' Bd. 99, H. 1.
(31) "Ueber die ätiologischen Beziechungen zwischen Syphilis und Tabes."—'Deutsche Med. Wochenschrift,' 1885, Nos. 1 and 2.
(32) "Hereditary or Degenerative Ataxia."—'Boston Med. and Surg. Journal,' 1885, vol. cxiii. p. 36. The last three papers mentioned will be found abstracted in the 'Neurologisches Centralblatt' for 1885.
(33) 'Amer. Jour. Med. Sci.,' April, 1885.
(34) 'Rev. de Méd.,' July, 1884, p. 555. Abst. in 'BRAIN,' Jan. 1886.
(35) 'Rev. de Méd.,' No. 2, 1886, p. 170.
(36) "Zur Casuistik der trophischen Störungen bei der Tabes Dorsalis."—'Berl. Klin. Woch.,' 1885, No. 12. Abst. 'Neurol. Central.,' 1885, p. 228.
(37) 'Amer. Jour. of Nervous and Mental Disease,' No. 2, 1885.
(38) "Iets over tabes dorsalis."—'Weekbl. van het Nederl. Tijdschr. voor Geneesk.,' 1884, No. 46. Abstract in 'BRAIN,' 1885, p. 86.
(39) "Ueber Vagus-Erkrankung im Verlauf der Tabes dorsalis."—'Berl. Klin. Wochens.,' 26 Jan., 1885.
(40) "Die Beziehungen der Hemikranie zur Tabes dorsalis."—'Berl. Klin. Woch.,' 22 Sept. 1884.
(41) "Tabes dorsalis."—'Rev. de Méd.,' t. v. 1885, p. 60.
(42) "Drei Fälle von Tabes im Kindesalter."—'Berl. Klin. Woch.,' 16 Feb. 1885.
(43) 'Le Progrès Médical,' 27 Oct. 1883.
(44) 'Path. Trans.,' 1884, p. 312.
(45) "Mollities Ossium, Rheumatic Arthritis, Charcot's Disease."—'Path. Trans.,' 1884.
(46) "Partial Mollities, Charcot's Disease, Spondylolisthesis."—'Path. Trans.,' 1884.
(47) "A Case of Charcot's Disease attacking the right elbow and foot."—'Clin. Soc. Trans.,' vol. xvii. p. 118.
(48) 'Clin. Soc. Trans.,' 1884.
(49) 'Clin. Soc. Trans.,' 1884.
(50) 'British Medical Journal,' April 4, 1885.
(51) 'Path. Trans.,' vol. xxxvi., p. 48.
(52) 'Path. Trans.,' vol. xxxvi., p. 352.
(53) "On Charcot's Joint Disease."—'Lancet,' July, 12, 1884.

(54) 'Cases of Joint Disease with Locomotor Ataxy."—'Clin. Soc. Trans.,' vol. xviii.
(55) 'Brit. Med. Jour.,' vol. ii. p. 886.
(56) "Ueber Kniegelenke Affection bei Tabes."—'Deutsche Med. Woch.' Dec. 10, 1885.

The recent advances in our knowledge of the Tabes Dorsalis, or Locomotor Ataxy, are in the direction the former advances have been in other diseases. Thus many years ago a doctor was content with the diagnosis of jaundice, as are the laity now; but at the present time the physician who did not diagnose the cause of which jaundice is only a symptom, would be looked upon as ignorant. In the same manner it is to be hoped that we are within measurable distance of the time when the diagnosis of a mere symptom, such as ataxy, will not do, but instead, we shall be able, from the grouping of symptoms, to say exactly what parts are affected. To a certain extent we are able to do so now; thus Pick (1) points out that we may have a cortical ataxy, a cerebellar ataxy, a spinal ataxy, and a neuritic ataxy. Still the phrase locomotor ataxy is usually reserved for the spinal form, and future investigation will be directed to discovering the exact parts of the spinal cord affected, and the reason why the patient should be ataxic when those parts are affected. Erb (2) has well pleaded for a more careful consideration of the facts of each case before adopting any theory, either Leyden's sensory one, Friedreich's motor one, or Strümpell's modification of the two. He throws out the suggestion that the ataxy must be due to some affection of that part of the motor tract, between the centres of will and the anterior cornua cells, which has to do with the co-ordination of movements. As disproving the sensory theory, he contrasts two cases, both of which were highly ataxic; but in one there was great sensory perversion, in the other none at all.

The best recent paper on locomotor ataxy, looked at from the point of view we are considering, is by Spitzka, (3) who says that the initial lesion is either in the posterior grey horn, or in that part of the column of Burdach traversed by the inner division of the posterior roots as they reach the level of entry into the grey substance. It is distinct from the secondary degenerations which accompany it. The affections of the columns of Goll and Clarke and the cerebellar tract are truly systemic; that of the middle area of the column of Burdach probably is not, whilst that of the root zones (*bandelettes externes*) certainly is not. The reasons for looking upon the degeneration of the column of Goll in tabes as a true secondary degeneration are, that when the lesion in the primary field is the lower lumbar or sacral, the degeneration in the column of Goll is limited to its postero-internal lower; when the primary disease is lower dorsal or upper lumbar, the rest of Goll's column is affected; and when the brachial, the comma-shaped area of Burdach's column just external to Goll's column, is affected; the degree of the affection of these areas bears a direct relation to the degree of motor ataxy. Thus, when there is motor ataxia of the arms, the comma-shaped area of Burdach is involved; when of all parts of the lower extremities, the column of Goll. Both the column of Goll and the direct cerebellar tract are under the trophic dominion of the posterior roots; thus the amount of degeneration in them generally corresponds, and hence static ataxy is found in advanced cases of tabes. Not only does the degree of the involvement of the column of Goll vary with the amount of motor ataxy, but it is unconnected with any other symptom, and affection of the cerebellar tract is connected with no other symptoms than static ataxy, and that of the column of Clarke is also connected with this symptom. It appears from several carefully recorded cases, that there is a small area with its base at the periphery of the cord, one side along the inner border of the posterior root for a short distance, and the remaining side joining the other two, which is affected in cases in which there are pains and hyperæsthesia, and profoundly diseased when there is analgesia, and in no case in which there was pain-disturbance was this area healthy. The affection of the bandelettes externes causes the abolition of the tendon reflexes. The extreme anterior part of Burdach's column is hardly ever diseased, and we have not at present been able to localise the lesion of disturbances in the tactile sense, or of sexual, bladder, rectal, or trophic troubles. The destruction of cortical control is inimical to the development of typical ataxy; hence patients who have early locomotor ataxy, and then develop cerebral symptoms, lose their ataxy.

Bechterew, (4) as a result of his observations on the development of the cord in the fœtus, comes to the conclusion that there are at least two distinct areas in Burdach's column, viz. one which stretches from the posterior root entry, along the periphery of the cord (it would seem probable that this corresponds with the analgesic tract mentioned above), and an anterior which lies along the inner border of the posterior cornua behind the posterior commissure.

Krauss, (5) as far as he goes, agrees with Strümpell in the position of the primary lesion, and the secondary lesions of Goll's column, as quoted above in Spitzka's article, which is founded partly on Strümpell's results. But here it may perhaps be pointed out that Spitzka totally disagrees with many of Strümpell's localisation of symptoms ; for example, the latter thinks that the bladder-disturbance is due to disease of Goll's columns ; but apparently this is totally incorrect, not only pathologically, but from a comparative anatomy point of view, for the porpoise, in which Goll's columns are very small, has no posterior extremities, although the bladder is developed as in other mammals. Krauss himself does not offer any views on the position of the symptoms in connection with the lesions, but records ten cases, to show that there may be ataxy without any change in grey matter, and that the primary seat in the root zone may be affected without any patellar reflex alteration, both of which statements are easily understandable.

As also tending in the direction of limiting more exactly the parts of the cord affected may be mentioned a short paper by Lissauer, (6) who describes an ascending bundle of fine fibres which are first on the inner side, later in the posterior horn. This bundle, he says, is diseased in locomotor ataxy.

Westphal (7) gives a very full record of a case. In the cervical region the inner part of the column of Goll and the boundary line between it and Burdach are affected, lower in the cord the latter gets nearer and nearer the posterior cornua. In the upper dorsal region it is triangular, whilst the Goll part is broader. In the mid-dorsal region both tracts are larger. In the lower dorsal and lumbar regions there is affection of the apex of the post-median fissure. In the cervical region the lateral column is slightly affected, in the lumbar regions the anterior root zones, and in the medulla oblongata the cuneate and slender fasciculi. Peripheral neuritis of the right lower extremity, and also changes in the roots of the spinal nerves, are present. Westphal attributes the non-abolition of the knee-phenomena to the root zone in the dorsal and lumbar regions remaining free. There was absence of ataxy, which he thinks remarkable, and thinks that probably the cause of this anomaly is to be sought in the intensity of the degenerative process. The extreme disturbance of sensation which was present was due to the peripheral neuritis.

Putzel (8) describes the histological appearances of a case; but from his description we cannot draw any conclusions as to the relation of symptoms to the disease of distinct strands, for all he says is that the extreme anterior part of the posterior columns was not affected (in this point he agrees with all other observers), the grey matter was normal, the change was least marked in the posterior root zones, and the posterior parts of the posterior columns; but there was a general increase of neuroglia in many parts of the cord. Post-mortem examinations on cases of tabes are few, and careful descriptions of the cord difficult to make; still it will be seen that all the recent literature goes to show that the posterior columns and posterior cornua are a very complex part of the cord, and contain many tracts. Only the most careful observation can show what symptoms are connected with what lesions, whether the various strands are affected by Wallerian degeneration secondary to affection of a primary field, or whether the disease is a diffuse sclerosis, attacking irregularly sometimes one and sometimes another band of nervous tissue in the posterior columns. The fact seeming to point to the last conclusion is the well-known one that the disease is very likely to occur before, and be followed by general paralysis of the insane (Savage, Insanity, and allied Neuroses), which is well known to be a diffuse disease. Baillarger (9) has recently published two interesting cases showing the association of the two diseases. Turner has published a case (10) in which the posterior columns, lateral columns, and grey matter were affected; and Ormerod (11) has brought up the knowledge of the association of disease of the lateral columns, with that of the posterior, to recent date. In all probability it will be found that the name locomotor ataxy, as the name of the disease, will have to be got rid of altogether; whilst the name tabes dorsalis might be retained for those cases in which there is primary lesion of the posterior cornua, with secondary Wallerian degenerations; whilst a whole mass of cases, some of which are peripheral neuritis, some are diffuse myelitis, and others a chronic meningitis, will have to be relegated to their respective positions.

The next pathological point which has been hotly discussed is whether the change is a primary neuritis, primarily a sclerosis of the interstitial tissue, or primarily a disease of the blood-vessels. Adamkiewicz, (12) as is well known, brought forward prominently the view that tabes was always due to primary sclerosis of the connective tissue. This was undoubtedly too sweeping an assertion. Schultze (13) has criticised his conclusions; a controversy sprang up between the two authors, and may be found in subsequent numbers of the same periodical. Still it is extremely probable that in many cases it is a primary sclerosis of the interstitial tissue, or perhaps in others a sclerosis due to a meningitis. As an example of this causation may be mentioned a paper by Déjérine (14). Here he records cases in which there was a posterior and lateral leptomeningitis, with a sclerosis of Burdach's and Goll's columns, and also a slight affection of the external part of the lateral columns. Krauss, in the article already quoted, does not incline to the view of a primary leptomeningitis. Ribail (15) records some cases of old people, in some of whom there was a sclerosis of the cord in connection with chronic meningitis, and bony plates in the membrane in the others; but whilst it is possible that the meningitis may have had something to do with the sclerosis, I think there is no doubt that the presence of the bony plates was only a coincidence, for they are so commonly observed without any affection of the cord. As far as I know, no very recent important work has been done with a view of determining which is really the more important factor—a parenchymatous or an interstitial neuritis; but several papers have appeared, discussing the probability of disease of the vessels being the primary lesion. Of these I think the best is one by Buzzard, (16) in whose case there was much inflammation of outer coat of the vessels of the cord, causing dilatation of their channels. The lesion was widely diffused. The posterior cornu, the cerebellar and crossed pyramidal tracts and anterior cornual cells were affected. The membranes of the cord were notably thick and adherent. Buzzard thinks that here the periarteritis was the primary condition. Demange (17) has recorded cases of the most marked kind in the spinal vessels of the old people, which, he says, were exactly like the changes seen in the cerebral vessels in old people, and which probably, through the medium of miliary aneurysms, lead to hæmorrhages. These changes, he thinks, lead to sclerosis of the cord, but Krauss (*op. cit.*) tested the question by seeing if there was any difference in the vessels in tabetic cases and in cases of secondary degeneration, and he could not find enough to warrant the conclusion that the changes in the vessels were primary in tabes. Demange, (18) soon after his last article, published another case in which the patient, 75 years old, had progressive contraction of the limbs, diffuse sclerosis of the lateral columns, generalised atheroma, periarteritis of the spinal vessels, granular kidneys, atheroma of the cerebral arteries, but no cerebral softening. The sclerosis affected slightly the columns of Goll and the anterior cornua, as well as the lateral columns. The author proceeds at great length to discuss all his cases, and concludes that atheroma of the spinal arteries will lead to sclerosis of the lateral columns, with spasmodic tabes, just as atheroma of the renal artery will lead to interstitial nephritis. But against his reasoning we may point out firstly, that it is by no means universally allowed that atheroma of the renal artery will lead to interstitial nephritis; secondly, that atheroma of the cerebral arteries does not cause sclerosis of the brain, but softening or cerebral hæmorrhage; thirdly, that it is extremely unlikely that atheroma of the vessels would give rise to a system lesion such as lateral sclerosis, for that seems to have been the predominating lesion, although the author calls his cases at the end of his article, ones of diffuse and disseminated sclerosis.

This is a good opportunity for pointing out that it has lately been urged that cardiac disease has some connection with ataxy. Grasset (19) refers to a short paper by O. Berger and O. Rosenbach (20). The only lesion observed by those authors was aortic disease. Grasset then records two cases that he himself has observed, and collects together a number from other authors. He concludes that valvular lesions other than aortic may exist, and that frequently the cause of the heart-disease is very obscure. He does not consider that the spinal cord and cardiac lesions stand in the relation of cause and effect to each other, but points out that the tabetic cases in which cardiac lesions are present usually have very severe pains, and suggests that, as it is well known that severe peripheral stimulation will affect the heart reflexly, it is possible that the cardiac disease may be caused in this way. I need hardly point out the extreme improbability of this suggestion; since the publication of Grasset's paper no other facts have been put forward to support it. Richardière (21) has published a case of tabes in which, with very peculiar trophic changes, aortic disease occurred. He is surprised that he cannot find any cause for the atheroma which caused the aortic disease, although he mentions the presence of syphilis in his patient. From reading the case through, one cannot help thinking that there is no need for him to draw attention to the fact of the association of aortic disease and tabes, considering that syphilis is assigned as a cause for both tabes and atheroma. We may, I think, conclude this part of the history of tabes with the pretty certain assurance that at present there is nothing to show more than an accidental connection between that malady and heart disease.

There has been much controversy as to the ætiology of tabes; and as is always the case when the exact limitation of a disease is vague, the opinions as to the causation are various. Until we can be sure of the exact pathological lesion in each of the cases, and all authors are agreed as to the pathological lesion which shall be called tabes, it is hopeless to attempt any certain knowledge as to causation; or at least the group of clinical symptoms to which the name tabes is given must be more exact than now—as exact, for example, as the group of symptoms constituting chorea; which, although it has no pathological basis, yet has a distinct ætiology.

To take an example; it is quite clear that all cases of peripheral neuritis must be excluded, and there can be no doubt that many cases that were formerly put down as locomotor ataxy, or tabes dorsalis, were in reality cases of peripheral neuritis. Perhaps the most instructive paper on this aspect of tabes is one by Dr. G. M. Hammond (22). He maintained that the disease could recover, but that the cases which did so were examples of posterior spinal congestion. In passing, one may remark that this at present is the purest hypothesis. In the discussion which followed on this paper, it was pointed out by several speakers that it was extremely probable that the cured cases were not posterior spinal sclerosis at all, but were other diseases, which at present it is very difficult to separate off during life from that malady. Thus Dr. Bartholow mentioned cases of mercurial ataxic and syphilitic gummata of the spinal cord which had been cured. Dr. Webber referred to a case of Déjérine's, in which during life locomotor ataxy was diagnosed, but the post-mortem showed peripheral neuritis. Now had this case been a recoverable peripheral neuritis, it would have been set down as locomotor ataxy recovered. One must also remember that syphilis and hysteria may cause symptoms which would very much resemble locomotor ataxy. Saturnine ataxy is also known; it is curable, but is probably a peripheral neuritis. Post-diphtheritic disorders of a rare nature might easily be mistaken for tabes dorsalis, as might the diseases kak-ke and beri-beri.

As an example of post-diphtheritic ataxy I may refer to a case by Berwald (23). The patient, three weeks after diphtheria, had paralysis of the palate, and soon after pains, deafness, difficulty of walking and using the hands. The author gives references to other cases, and, from an analysis of the symptoms in this one, comes to the conclusion that it is not a peripheral neuritis.

Then again there is Hughes Bennett's case (24), in which there was no disease of the posterior columns, but a number of tumours on the posterior roots, and sarcomatous softening of the medulla, and yet the case was diagnosed during life by every one who saw it as one of locomotor ataxy. Dr. Bennett has carefully considered the cause of the absence of medullary symptoms, and hence I do not think that Spitzka's (*op. cit.* p. 395) criticism is valid.

A case of saturnine ataxy will be found recorded at the same meeting of the American Neurological Association as that at which Dr. Hammond's paper was read.

Then also there is the question whether in papers dealing with the ætiology of tabes, cases have been excluded in which the tabes was either accompanied by, or secondary to, other diseases of the nervous system. I have already referred to the fact that it often precedes or follows general paralysis, which is well-known to be due to syphilis. Some marked cases showing this association have been recorded by Seguin (25) and by Fournier (26), and Camuset (27) has recorded a case in which without ataxy there was primary sclerosis of the columns of Goll in an old person who was mentally weak.

In the number of 'Brain' for January, 1886, will be found abstracted a case by Sakaky, in which peripheral neuritis existed in association with locomotor ataxy, and it is pointed out that several other such cases have been recorded; for example, Krauss (*op. cit.*) mentions some. All these references show the difficulty of separating tabes exactly from various diseases of the nervous system, and hence of discovering its cause.

Belugon (28) discusses at some length, and by the help of elaborate tables, the cause. With regard to syphilis, he points out that the existence of the two diseases is often only a coincidence, because anti-syphilitic treatment did no good to the tabetic symptoms. He concludes that syphilis rarely causes the disease, but that it puts the patient in such a condition that he is very likely to be affected by tabes. Hereditary influence is of some ætiological value, but not of so much as syphilis. Rheumatism is of all causes by far the most important. He is not able to decide upon the value of excesses as a cause, but considers that sexual excess may be one. In a subsequent number of 'Le Progrès Médical' there is a letter from MM. Landouzy and Ballet, to point out that they have also recognised the importance of rheumatism.

It is well known that some authors have pointed out the importance of syphilis in tabes. Erb (29) brings forward 100 cases of tabes, 91 of which had syphilis. The percentage of syphilis among non-tabetic cases at the same clinic being 22·75. Also he says it is noteworthy that tabes usually occurs late in life among those who have acquired syphilis late. Again, in both diseases the proportion of men to women is ten to one, and both are common in the lower classes, rare in the upper. Since the publication of that paper, Eulenburg (30) and Berger (31) have pointed out the strong association between the two diseases. Eulenburg's percentage is not quite so high as Erb's. Berger gives some interesting cases; the most striking is that of a man who, at the age of 68, acquired syphilis, and at 72 years old developed tabes. The percentage in this paper is pretty much the same as that in Eulenburg's. The hereditary influence is well shown in some cases published by Everett Smith (32), who gives an account of a family of thirteen children, of whom five had ataxy, and the disease was also present in the father. It will be seen that at present, syphilis, excesses, hereditary influence and rheumatism, seem to be of some ætiological value, but how much cannot be determined till tabes is more exactly defined.

From time to time rare symptoms are recorded as occurring in the course of tabes. In fact they are often so rare, that one cannot help thinking that perhaps their presence is a mere coincidence. A few of these more unusual accompaniments are here recorded.

Birdsall (33) gives an account of some of the ocular affections. It appears that visual acuteness is nearly always unequal in the two eyes, but patients retain longer the power of seeing near than far. Unusually strong electric currents are required to produce a sensation of light. Hyperæsthesia and anæsthesia may be observed over the distribution of the 5th nerve, the former being accompanied by excavation of the optic papilli. There may be colour-blindness.

G. H. Roger (34) says that out of 32 cases he examined, 14 had diarrhœa, and in 5 of them there was reason to think it had special connection with the disease. It may or may not be accompanied by abdominal pain. It is often sudden, and the evacuations are watery.

Many of the symptoms recently recorded have been of a trophic nature. Richardière (35) has recorded a case of disease of the metacarpo-phalangeal joint, with luxation of the thumb in locomotor ataxy. In the same case the teeth fell out. The patient thus describes this occurrence. A year before admission into the hospital, the eyes and face swelled, and one by one the teeth fell out without any caries; after this a few pieces of bone fell out from the palate. On admission to the hospital it was discovered that the gums were healthy, that the upper teeth had gone, but that the lower were all there.

Hoffmann (36) records three cases of tabes. In the second, without any cause, the patient, a man of 48, lost in about ten days all the teeth of the upper jaw. Two years after, the tabetic symptoms began. The first case, that of a man aged 59, is peculiar from the fact that a subcutaneous rupture of the tendo Achillis took place spontaneously. This is an excessively rare trophic lesion in tabes. Dr. Lewis (37) and other authors have also described cases in which the teeth fell out.

Unusual trophic changes in the form of grey hair and pigmentary changes over the 5th nerve distribution will be found in a paper by Klinkert (38).

Oppenheim (39) has published a very interesting account of laryngeal crises, and after referring to the fact that sometimes the vagus or recurrent laryngeal has been found affected atrophically, and that it would appear that the symptom may be due to disease of the 4th ventricle, he records a case of a man who had peculiar crises accompanied by dyspnœa. On admission he had a falsetto voice. On deep inspiration there were present much difficulty of breathing and stridor, paralysis of both crico-arytænoidei postici and the right lateral crico-arytænoid, difficulty of swallowing, pain on pressure at the inner border of the sterno-mastoid, and temporary failure of pulse with attacks of giddiness. Then the author gives an account of a patient with very similar symptoms, in whom was found degeneration in the vagal trunk and the recurrent laryngeal. The same author (40) points out the frequency with which hemicrania is associated with tabes. Out of 85 cases he found that 12 had had typical migraine, and of these 12, ten were men and two were women.

Vulpian (41) describes a case which progressed slowly, and was verified by a post-mortem examination. The patient in 1878 was seized with violent epigastric pain, diarrhœa, and vomiting; from this he recovered and remained well two years, when he had a similar attack. He went in and out of the hospital several times, but it was not till 1882 he was finally admitted, when tabes dorsalis, with gastric crises and angina pectoris, was diagnosed. He had repeated attacks of cardiac pain, the association of which with tabes forms the chief interest of the case; he died at last of phthisis.

It is well known that, putting aside cases of Friedreich's disease, cases of ataxy are not common in young children; but Remak (42) gives three cases of various tabetic symptoms in children who had had congenital syphilis and had optic atrophy, one was 12, another 14, and another 16 years old. They were no relations to one another.

Ballet (43) has pointed out that hemiatrophy of the tongue is not very infrequent. It may occur on either side; the affected side is thrown into wrinkles and furrows. There is sometimes a slight contraction with vermicular movements; it points to the affected side; it is of a very early symptom, and produces no effects on deglutition, mastication, or speech. Since this paper, other cases have been recorded.

In England, discussion about Charcot's disease has waxed very warm during the last two years, and although every one has had an opportunity of expressing his opinion, it is doubtful whether our exact knowledge of the subject has been much advanced. If this be a genuine symptom or complication of tabes, it labours under the disadvantage that probably many of the cases in which it exists have not been tabes at all, but peripheral neuritis, or some other disease closely resembling tabes.

In November 1883 I showed (44) a pelvis which, although no history could be obtained, for it was a dissecting-room specimen, was so like to those specimens described by Charcot as characteristic of tabes, that I got my friend Prof. Féré to show it to Prof. Charcot, who pronounced it to be an example of the disease named after him. It certainly is the most unusual pelvis I have ever seen. It weighed but 7 oz. The bones were extremely thin, this being best shown in the acetabula, which have been pressed in to form two great bosses on the interior of the pelvis. Owing to this deepening of the acetabular cavity, the lesser trochanter had worn away a place for itself at the margin, and thus the cavity was much larger than natural. At the margin of the cavity there was the merest trace of new bone. The specimen is in the Guy's Hospital museum. At the conclusion of my paper, my doubts are expressed whether Charcot's disease is a distinct pathological entity.

Mr. Arbuthnot Lane (45) pointed out that the joints affected in rheumatic arthritis are also affected in Charcot's disease; that the absence of osteophytic growth is not a point of distinction; and finally, Mr. Lane could not find any points of distinction between the two diseases, and considers that when rheumatic arthritis occurs in old people it presents such degenerative changes as to be indistinguishable from Charcot's disease, which, when it is found in strong healthy people, is indistinguishable from rheumatic arthritis. Thus they are one and the same disease, modified by the patient. The same author (46) mentioned a pelvis deformed in a very similar manner to others previously described, and again emphasized his opinion that Charcot's disease is rheumatoid arthritis, modified by the degenerated condition of the tissues involved. Mr. Clement Lucas (47) gave the case of an ataxic man in whom the elbow presented many bony prominences and some crackling; the right foot was swollen, but neither tender nor œdematous, although painful; but Mr. Lane (*op. cit.*) thought this case was one of rheumatic arthritis, and I must say that, on examining the patient, I thought so too. Dr. Buzzard showed (48) a case of Charcot's disease in the shoulder, and it certainly corresponded much more closely to Charcot's description than did Mr. Lucas's case. Cases of perforating ulcers of the foot in locomotor ataxy have been recorded by Dr. Duckworth (49) and by Dr. Suckling (50). Dr. Paddison (51) mentions a case of Charcot's disease. Examination of the spinal cord showed affection of the columns of Goll in the crural region and the posterior columns in the lumbar, and Mr. Hopkins (52) describes two cases of arthritis occurring in locomotor ataxy. I collected (53) the evidence for and against regarding this disease as a distinct affection, and pointed out that Mr. Hulke, Dr. Moxon and Dr. Goodhart had in discussions at the societies all expressed themselves in favour of the view that it was a modification of rheumatoid arthritis. I enumerated three views which might be held: first, that the joint-disease is a distinct specific arthropathy peculiar to locomotor ataxy; against this it may be said that although the anterior cornua, the medulla and the peripheral nerves have all been said to be the cause of the arthropathy, yet it is highly probable that none of them can be looked upon as constant causes; then with regard to the clinical symptoms, a large number of observers know nothing of the most characteristic symptoms described by Charcot, but recognise the cases as examples of rheumatic arthritis, and many of Charcot's symptoms are also symptoms of rheumatic arthritis; out of six cases of joint-trouble with locomotor ataxy I have seen, there was not one which all observers agreed was rheumatic arthritis, nor one which they all agreed was Charcot's disease. The morbid anatomy of the English specimens is discussed, and also Charcot's account of the morbid anatomy of his, and it is shown that there is no point distinctive of the two diseases; and the case is mentioned of a man whose larger joints presented all the rheumatoid arthritis changes, whilst the temporo-maxillary articulation was exactly like Charcot's description. The second view is that the joint-disease of locomotor ataxy is really the same thing as the joint lesions produced by injuries to nerves, or due to cerebral hæmorrhage. It is quite possible that some of them may be of this nature; but if so, why not at once put them in the same class? References to the literature of nervous arthropathies are given. The third view is that it is really an ordinary arthritis occurring independently of tabes, but modified by the conditions which it enforces. Many arguments, such as that of the lowered vitality, the prolonged rest in bed, the liability to traumatism, are brought forward for this view, and it is urged that no one has ever shown that ataxics are more liable to joint-disease than other people, which they would be if there were a specific arthropathy, for then they would be liable to that in addition to their liability to rheumatic arthritis.

In Mr. Morrant Baker's (54) paper the first case is that of an ataxic woman, in whom there was flail-like movement of hips and elbow, with bony bosses and grating in the latter; the right metatarso-phalangeal great-toe joint was also affected. In the second case the right knee was affected, and there was some dead bone in the right toe. Post-mortem examination showed sclerosis of the columns of Goll and much increase of bone in the neighbourhood of the knee. In the third case the knees were affected, and both during life and at the post-mortem showed most of the changes described by Charcot, and also some points of resemblance to rheumatoid arthritis. Mr. Morrant Baker came to the conclusion that it was "only in an exaggerated form what had long been familiar to us under the name chronic rheumatic arthritis." He thought the association of tabes and joint-disease was not a mere coincidence, and after rejecting the neurotic view of chronic rheumatic arthritis, he went on to say that it seemed to him "conceivable that the ataxy and joint-disease might be due to some pre-existing depraved condition common to both of them," and in proof of that view he refers to a well-known letter by Dr. Wilks (55), which points out that both pathologically and clinically at present we have no definite disease tabes dorsalis. In the discussion which followed the reading of Mr. Baker's paper, Mr. Barker showed a specimen of the disease. Dr. Dyce Duckworth thought Charcot's position proved; Dr. Hale White considered most of the cases were modified forms of rheumatoid arthritis; whilst Dr. Buzzard and Mr. Barwell agreed with Dr. Duckworth. Sir James Paget thought that in a certain sense it must be looked upon as a new disease, for in the collections of the older observers no specimens could be found. The fact that we named it after Charcot showed we knew very little about it pathologically. "It might well be a rheumatic arthritis modified by its coincidence with disease of the spinal marrow." The extremes were distinct; but as they ran one into the other at the borderland, it might justly be suspected that the diseased joints were really examples of chronic rheumatic arthritis. The next speakers, viz. Dr. Ord, Prof. Humphry, and Mr.

Hutchinson, in the main agreed with Sir James Paget. Mr. Halke criticised Mr. Barwell severely, and felt quite unable to agree with those who looked upon Charcot's disease as a distinct thing from rheumatoid arthritis; in this view Dr. Moxon also most fully agreed. Mr. Morris showed two interesting cases; one of disease of the knee, and another of disease of the ankle, which could not be distinguished from Charcot's disease. The patients were without ataxy. In the second case there was elephantiasis of the leg and perforating ulcer, together with the nerve changes usually found in that disease. Mr. Page, Mr. Macnamara, and Mr. Clement Lucas believed in Charcot's disease as a distinct disease, whilst Dr. Pye Smith did not. Many other speakers spoke; but enough have been referred to show that on the whole the opinion was distinctly in favour of Charcot's disease being a modification of rheumatoid arthritis. Mr. Baker, in his reply, reiterated this view. A letter was read from Prof. Charcot, who expressed his surprise that this was the prevailing opinion in England.

Leyden (56) is very undecided as to the relation of tabetic joint-affections to rheumatic arthritis. On the other hand, at a meeting of the Philadelphia Neurological Society, Jan. 26th, 1885, which was reported in a subsequent number of the 'Journal of Mental and Nervous Diseases,' Drs. Wood, Roberts, and others, read papers on Charcot's disease; the prevailing impression was that it is a distinct disease.

From this review of our knowledge of tabes it would appear that we have not advanced much, but in a way we have, towards the time when one or many distinct diseases with distinct pathologies and distinct symptoms will crystallize out of the mass of conditions now grouped together under the name tabes dorsalis.

ON THE ETIOLOGY OF TABES DORSALIS.

BY JAMES PIETERSEN.

UNDER the title of "Over de Aetiologie der Tabes Dorsalis." Dr. B. H. Stephan contributes to the 'Nederlandsch Tijdschrift voor Geneeskunde' an interesting paper, summing up all we know from Continental writers on the subject, and raising the interesting question whether Locomotory Ataxy cannot be regarded rather as an affection of the whole cerebro-spinal system, due directly to change in the vessels supplying the nervous system, than as has heretofore been done, attributing all the symptoms to one or more definite cord lesions; and among the causes which favour changes in the blood-vessels he gives syphilis the first place, making it thus indirectly the main, though not the exclusive cause of tabes. Regarding sexual excess as a primary cause, he says, "One can well understand that sexual excess, even as a severe attack of typhus, syphilis, alcoholism, rapidly following and more or less continuous lactation, may now and then favour the development of this affection; but having regard to the relative frequency with which sexual excess is met with, and the relative infrequency of occurrence of tabes dorsalis, it seems to me that the assumption of such as a primary cause is wholly unwarrantable." Exposure to cold, as well as mental and physical strain, he regards, with hereditary predisposition, as of secondary moment. He then quotes Fournier as the earliest promulgator of the syphilitic origin of tabes, whose disciple Erb went so far as to affirm that no one could evince ataxic symptoms without being able to account for a previous syphilitic affection. "The main arguments brought forward by those who deny the syphilitic origin of locomotor ataxy are to be classed under three heads.

"1. The almost inappreciable success of specific treatment.

"2. That the lesions in tabes, limited as they are to certain anatomical tracts, in no way correspond with other syphilitic affections of the nervous system.

"3. The unreliability of the statistics brought forward."

Putting on one side the first two opposing arguments, which he deals with more fully in the body of his paper, he holds that statistics are of great value in the consideration of this question, and that there are three points for us to consider when we look at it statistically.

"(1.) Are the numerical results in the various tables writers have drawn up to show the connection between syphilis and tabes proportionately alike?

"(2.) Do investigators regard the early manifestation of a chancre of doubtful character, or a soft sore, as sufficient warranty that when tabes occurs a syphilitic cause should be assigned to the malady? [for the distinction between the hard chancre and soft sore, in that they are each due to a separate and distinct virus is by no means universal].

"(3.) Is the tabulation of such cases where syphilis preceded tabes satisfactory, when a doubtful chancre or a soft sore was considered sufficient evidence to assume syphilis?"

Notwithstanding these weak points, he holds that a remarkable relation exists between tabes dorsalis and earlier syphilis, "for when Bernhardt computes that of 125 ataxic patients, 46·8 per cent. had some syphilitic antecedents, while of 155 others, not ataxic, only 15·8 per cent. had had syphilis (in both cases soft chancre being regarded as non-syphilitic), then undoubtedly some connection must exist between these two affections." He goes further, quoting *Landsberg*,[1] who published a case of locomotor ataxy with precursory syphilis, where the nerve affection yielded to a specific treatment, mainly inunction, and where the malady at the time of publication had almost wholly disappeared, the most undoubted ataxy having manifested itself previously: *Berger*,[2] who relates the history and post-mortem appearances of a case that came under his observation, where syphilis was contracted in the 69th year, followed by slight secondary symptoms. Three years later double interstitial orchitis, with typical tabes, manifested themselves; the diagnosis was verified by the autopsy: *Voigt*,[3] too, draws attention to the access of tabes in old persons where syphilitic infection had taken place in the later years of life.

He does not, however, agree with Erb in giving syphilis as the exclusive cause of tabes dorsalis, for *Eulenburg*[4] could only prove precursory syphilis in 36·8 per cent. of his cases, Berger only in 43·8 per cent. "There are therefore," he continues, "in the development of this spinal affection other causes also at work. What these causes can be, and what the nature of the connection is between syphilis and these causes on the one hand, and tabes on the other, we must follow more closely."

He first of all draws attention to the action of poisons on the system, quoting Strümpell, that observation of such action often is of etiological value as regards cord affections. "We know that certain poisons act only on certain portions of the spinal cord, disturbing certain physiological centres without in any way affecting the whole cerebro-spinal system (instance the action of *strychnine* on the reflex centre: *curari* on the motor end-plates of peripheral nerves: *morphine* on the sensory central organs: *atropine* on the vasomotor and secretory centres). The action of certain poisons, too, is not alone the cause of functional, but also sometimes of anatomical change (e.g. *Plumbism* causing affection of the motor portion of a certain nerve: *Ergotism* evincing not only a clinical but also a pathological likeness to tabes)."

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[1] 'Berl. Klin. Wochenschrift,' 1885, No. 33.
[2] 'Deutsch. Med. Wochenschrift,' 1885, Nos. 1 and 2.
[3] 'Centralblatt für Nervenheilkunde,' 1885, No. 8.
[4] Virchow's 'Archiv,' Bd. 99, 1.

"More and more probable does it then appear," he continues, "that infectious vessel-affection (one species of poison-influence) plays etiologically a more important part in lesions of the spinal cord than up to the present it has been accredited with. Various writers have attributed some forms of spinal-cord sclerosis to primary arterial affection, and others have noted endarteritis as a coincident phenomenon in affections of the cord, without going more deeply into the question of the connection between them."

He then quotes *Letulle* and *Martin*, who have published cases where tabes was accompanied by general atheroma, and who mooted the question whether any connection can exist between the vessel-affection and the cord lesion: *Ballet* and *Minor*,[1] who published a case in 1884 of diffuse sclerosis of the lateral and posterior columns, where in the sclerosed portions endo-periarteritis plainly existed; the patient had previously been syphilitic. After that Ordonez had claimed to find in arterial change the primary commencement of nerve degeneration in tabes, Adamkiewicz has more recently[2] reasserted his view, the degenerated tracts in the posterior columns in tabes corresponding to the vessels affected. According to his ideas, the vessel affection in these cases would not act in a direct manner (and in this particular he differs from Ordonez), but indirectly through the medium of the interstitial connective tissue binding these vessels together, serving thus to bring about sclerosis and atrophy of the nerve-tissue. *Rumpf*[3] also, taking note of the thickening of the media and adventitia, considers arterial change in certain cases the primary cause of tabes. *Krauss* too[4] investigated in 13 cases of tabes the condition of the vessels in the posterior columns; in most cases thickening of the vessel-walls existed. No distinction could be drawn between such condition of vessels in tabes of syphilitic and of non-syphilitic origin.

"Affections of the heart, and notably of the aorta and aortic valves are not unfrequent in tabes. *Berger* and *Rosenbach* have published the first seven cases of locomotor ataxy with aortic incompetency. *Letulle* and *Grasset* have also collected some cases exhibiting the same association. *Tessier* thinks[5] that in many cases the aortic lesion is not diagnosed during life, and that, post-mortem, small perforations of the valves are to be found; more recently he has published a case where a patient with initial ataxic symptoms developed, after a long tramp one cold night, an aortic incompetency. *Déjérine*[6] described a case in 1884 which had been taken for one of amyotrophic lateral sclerosis, wherein, post-mortem, multiple patches of sclerosis were found, and in which also periarteritis existed. *Demange*[7] also published a case in that year which was considered one of amyotrophic lateral sclerosis, and in which a disseminated myelitis of vascular origin was found; endo-periarteritis of the vessels of the medulla, with general arterial atheroma, was likewise discovered. This year two cases appeared under his observation, in which there was found, post-mortem, a general atheromatous endo-periarteritis of the cord, and in which, during life, the symptoms of lateral sclerosis had existed. *Marie*[1] also has recently drawn attention to cases cited by *Westphal* of ataxy following acute infectious disorders, and has tabulated cases of multiple sclerosis following typhus, small-pox, erysipelas, pneumonia, measles, scarlatina, whooping-cough, intermittent fever, dysentery, diphtheria, cholera, and puerperal fever." He further speaks of arteritis and periarteritis (probably resulting from bacteria emboli) in their connection with the multiple patches in the brain and spinal cord, to define in conclusion such cases as cerebro-spinal localisations of an infectious vessel-lesion. "Acute poliomyelitis, acute encephalitis, and multiple neuritis, by their peculiar symptoms and course, cause us to suspect the influence of infection, wherein the infecting virus is localised in the cerebro-spinal system, just as the virus of cholera is localised in the intestinal tract;" and he quotes cases contributed by *Mulder*, which go far to prove that such a surmise is a correct one.

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[1] 'Archives de Neurologie,' 1884, No. 7.

[2] 'Die Rückenmarksschwindsucht,' 1885.

[3] 'Archiv für Psychiatrie,' Bd. 15, No. 2.

[4] 'Neurologische Centralblatt,' 1885.

[5] 'Gazette hebdomadaire,' 1884, No. 40.

[6] 'Revue de Médecine,' 1884.

[7] 'Ibid.

"While observers then at the present day regard vascular change as the primary anatomical cause of various cord affections, there is but little unanimity when such a cause is assigned to tabes dorsalis. Others, opposing the idea that the initial changes arise from the vessels, consider a primary degeneration of nerve tissue as the anatomical starting-point—the doctrine, however, of vessel change as the primary anatomical cause of tabes is steadily gaining ground. Such being the case, and innumerable instances warrant the adoption of such an opinion, there can be no improbability in the assumption that affections which bring about endo- or periarteritis can cause by a spinal or cerebral localisation an acute or chronic change in the cerebro-spinal system. Among the influences which can occasion such a condition, syphilis stands prominently and undoubtedly at the head; but we also know, and *Duplay*[2] has lately again drawn attention thereto, that rheumatism, gout, chronic alcoholism, malaria and lead-poisoning, can bring about like changes; and it is also manifest that sometimes, after an acute infectious malady, endo-periarteritis of spinal vessels may ensue. It is possible that particular infectious vessel-affections such as these may bring about particular spinal cord lesions, as, for instance, that syphilis mainly induces changes resulting in tabes, and that causes other than syphilitic may bring about other spinal affections.

"But it must be remarked, in the first place, that undoubted cases of tabes can be cited without any precursory syphilitic affection, and that syphilitic and non-syphilitic tabes are indistinguishable, either pathologically or clinically, so that there can be no doubt that vessel-affections of non-syphilitic origin must also rank as responsible agents in bringing about locomotor ataxy; and also is it well worth considering more closely in how far from the pathological investigations, or from the symptoms of the disorder which we call tabes dorsalis, the localisation in a well-characterised anatomical or physiological manner of a certain poison can be determined." Reviewing then the rise and progress of our knowledge of spinal affections, through the combined aid of pathological research and clinical observation, he more particularly deals with the lesions found in tabes dorsalis. "But," says he, "it is not only to the degeneration of one definite column in the spinal cord that we can assign the lesion of tabes. The almost constant affections of the cerebro-spinal system in cases of tabes are:—

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[1] 'Progrès médical,' 1884.

[2] 'Archives générales de Médecine, 1885.

1. Changes in the meninges (so decided sometimes that some writers, *Arndt*, *Waldman*, and others, have endeavoured to show that tabes is preceded by a primary leptomeningitis).
2. Lesions of the posterior columns.
3. Lesions of the posterior roots.
4. Lesions of the grey matter of the cord.
5. Lesions of the cerebral nerves.

"Less constant, though still frequently observed, are:—

1. Changes in the cortex cerebri.
2. Changes in the sympathetic system.[1]
3. Changes in the spinal peripheral nerves."

In tabes, then, there can occur certain definite affections of the brain, spinal cord, and peripheral nerves; in other words, changes in the whole cerebro-spinal system. Various combined lesions also sometimes appear, sometimes of posterior and lateral columns,[2] sometimes of posterior columns and anterior cornua.[3] He further maintains that tabes dorsalis can exist while "the classical '*symptômes tabétiques*' of Charcot," with which the lesion of one definite portion of the posterior columns has been associated, are in abeyance. "Sclerosis of the posterior columns is indeed one of the evidences of this affection, but the diversity of the anatomical changes debars us from looking on this one condition as the pathological substratum of this nervous disorder." Clinical evidence is equally indefinite, the symptoms being neither regular nor constant. "*Fournier*[4] evidences psychical disturbances (loss of memory, change of character and disposition, and intellectual disturbances), especially in syphilitic tabes, as the forerunner of locomotor ataxy; with others, retinal atrophy of many years' standing is instanced as a precursor; some cases show disturbances of vesical functions; others gastric and other crises; others, again, epileptiform attacks as the antecedent symptoms. The absence of knee-jerk, a phenomenon first studied by Erb and Westphal, associated with degeneration of the posterior column, was regarded almost as a pathognomonic sign of the presence of tabes. The value of this as such has of late necessarily declined, now that the investigations of *Bernhard*[1] have taught us that after diphtheria this absence of knee-jerk is commonly observed; while later still, we find the same phenomenon noted in sufferers from chronic alcoholism, and in those in weak and debilitated conditions of the system; and still more recently the publications of *Bouchard*, *Rosenstein*,[2] and *Maschka*[3] have proved that in diabetes the same absence of knee-jerk is observable."

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[1] Raymond and Artaud

[2] Kahler, Prévost, Raymond, &c.

[3] Charcot, Leyden, &c.

[4] 'L'Encéphale,' 1884.

"In conclusion," he remarks, "what likeness can we draw between the pathological lesion and the clinical signs, when we find cases recorded where pains in the legs, anæsthesia, analgesia, Romberg and Westphal's symptom, ataxy, &c., were observed, while on post-mortem an intact spinal cord, with however a widely disseminated degeneration of peripheral nerves, was found (cases of multiple neuritis or neurotabes peripherica—Déjérine, Hirt, Hun, &c.), and where cases of diabetes are cited, which by their multiple nervous disturbances and evident absence of knee-jerk are to be regarded rather as cases of tabes (the urine not having been examined) than as diabetic, especially when we consider that cases do occur in which tabes dorsalis is accompanied by glycosuria (Althaus)? Regarding then the manifold changes met with post-mortem in tabes, the variety of the symptoms, their irregular grouping and inconstant exhibition, I believe it to be more consistent to designate those symptoms which we call tabes dorsalis, and which we have heretofore attributed to a lesion of one definite anatomical tract in the spinal cord as a universal affection of the cerebro-spinal system, and that in no instance can either pathological investigation or clinical evidence justify us in definitely attributing to syphilitic influence alone the lesion we find in the posterior columns of the cord." In conclusion, he formulates the opinions he has advanced above under four heads.

"1. Cerebro-spinal localisation of infectious vessel-lesions plays an important part in the etiology of affections of the brain and spinal cord (with which, too, particularly tabes).

"2. Syphilis undoubtedly leads most frequently to affections of the vessels; and, as tabes is the most frequent of spinal-cord maladies, it is only natural that syphilis should be a frequent precursor of tabes.

"3. That syphilis should be considered as the exclusive precursor of tabes, and no other chronic spinal affection is as unlikely as that excluding the syphilitic cause, no other malady causing vascular affection can pose as the cause of tabes.

"4. The direct reason why infectious vessel-lesions should at one time induce diffuse changes, at other times more or less plainly characterised systematic affections, is at present beyond our comprehension."

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[1] Virchow's 'Archiv,' Bd. 99.

[2] 'Berl. Klin. Wochenschrift,' 1885, No. 8.

[3] 'Wiener Medicinsche Presse,' 1885, No. 3.

Reviews and Notices of Books.

L'Encéphale, structure et description iconographique du Cerveau, du Cervelet et du Bulbe. By E. Gavoy.

This work consists of two parts, a series of plates of the human brain, and full explanatory text describing the various plates.

The text commences with a preface by Prof. Vulpian, who speaks in highly eulogistic terms of the importance and usefulness of the work, and commends alike the accuracy of the drawings and the amount of labour that it must have entailed on its author.

The first part of this text is devoted to a short general description of the nervous system, while the second part gives a special description of the grey and white matter of the brain. Under the description of the grey matter are given the cortex—with an account of its different layers and their constituent cells—and the basal ganglia. The white matter is described under the headings of arciform, commissural, and convergent fibres. The white and grey matter of the cerebellum and the medulla are likewise described. About forty-eight pages are taken up with this general and special account of the nervous system, while the rest of the text, over one hundred pages, is devoted to the explanation of the different sections.

The essential part of the work, the atlas, consists of a series of plates of sections of the human brain in different planes. The sections have been drawn by the author the exact size, and his drawings have been reproduced by the process of glyptography by M. Silvestre. Some idea of the magnitude of the work can be gained by the fact that only the thickness of one millimetre intervenes between each section and the succeeding one, and that altogether as many as fifty-five plates are given.

The method employed was as follows:—sections were made, one millimetre thick, by a cerebrotome in three different planes; they were then numbered and transferred to a bath, which did not alter the size or normal colour of the sections, but only made them more transparent, and they were mounted between two plates of glass which were sealed at their edges.

The first series, consisting of thirteen plates, was cut vertically, and from before back, *i.e.* sagitally; the second horizontally through both lobes of the cerebrum; while the third series, twenty-three in number, was made in a vertical transverse direction, *i.e.* frontally.

The drawings are most beautifully done and reflect great credit on the author, and the way in which they have been reproduced by glyptography by M. Silvestre leaves nothing to be desired. One would have liked to know the manner in which the brains were prepared, and the form and construction of the cerebrotome by which the sections were made. The author states that the sections were made when the brain was fresh, immediately after its removal from the cranial cavity, but he does not mention whether the brain was frozen or not, when the sections were made. Each section, as it was cut, was transferred to a bath, which, while it increased the transparency of the sections, did not in any way alter their normal colour or produce any diminution in size. The nature of this bath is, unfortunately, not described; and we think this is an important omission, as it would be very interesting to know by what means the author has been enabled to bring out the direction of the fibres very much more distinctly and definitely than it is possible to see in a section of a fresh unprepared brain. It would here be a fit place to mention that a similar atlas to the present one has been previously brought out in America by Prof. Dalton. This atlas consists of a similar series of sections of the human brain, taken in different planes, but with this great difference: that the brains are frozen, and the sections are photographed without any previous treatment or method being employed to bring out the appearances of the various structures. The difference in the number and appearance of the fibres in the drawings of Dr. Gavoy and in the photographs of Prof. Dalton is very marked, the fibres being more minutely delineated in those of the former, though we cannot but think that the latter must be the more accurate.

Though the drawings are most beautifully executed, we find ourselves unable to agree with some of the statements contained in the second part of the text, or the special description of the grey and white matter. On p. 17, § 3, a description of the converging fibres of the brain is given; and although the description occupies over three large pages, no mention is made of the pyramidal fibres, *i.e.* fibres passing direct from the cortex through the internal capsule to the pyramidal tract in the pons, without being attached to the optic thalamus or corpora striata. All the fibres are described as passing from the cortex to the basal ganglia, under the two heads of cortico-thalamic and cortico-striate. We think that even if the author does not believe in the existence of these fibres, that some mention of them should have been given. We must also take exception to the description of the fibres in the cerebellum on p. 25. It is there stated that the medullated fibres enter the grey matter of the cerebellar cortex, and they lose their medullated sheath, traverse the deep layer, and, reduced to their axis cylinders, they reach the superficial zone and there join the prolongations of the large and small cells. According to this account, the fibres lose their medullated sheath before traversing the deeper or granule layer. This, in our opinion, is certainly contrary to the facts of the case, as by using Weigert's method, the medullated sheath is seen to be present even as far, superficially, as the molecular layer, and as far as we know, no one has demonstrated the point where the medullated fibres lose their sheath. Although the medullated fibres join the basal side of the Purkinje's cells, no one has satisfactorily traced any of these fibres to the smaller cells of the molecular layer, though according to some observers these cells are joined by the protoplasmic processes of Purkinje's cells, but it is mere conjecture whether these cells are joined by medullated fibres from the granule layer.

The atlas will be of great use to any one wishing to learn the relations of the deeper parts, especially, of the cerebrum when viewed in different planes.

C. E. Beevor, M.D.

Abstracts of British and Foreign Journals.

Neurological Contributions from St. Bartholomew's Hospital Reports, 1874–1884:—

1874.

1. On the Clinical Value of Expression in the Insane. By T. Claye Shaw, M.D.

1875.

2. On Laryngismus. By Samuel Gee, M.D.
3. On Incontrollable Impulse. By T. Claye Shaw, M.D.
4. A Case of Idiopathic Tetanus resulting in Cure. By George Hastings.

1876.

5. On Phrenitis Æstiva. By Samuel Gee, M.D.
6. A Contribution to our Knowledge of the Physics of the Cerebral Cortex. By W. Ainslee Hollis, M.D.
7. The Histology of Certain Forms of Degeneration of the Tissues of the Nerve Centres. By W. Henry Kesteven.
8. Epidemic Cerebro-spinal Meningitis. By Neville Hart, M.B.

1877.

9. Further Histological Researches on the Pathology of the Nervous System. By W. Henry Kesteven.
10. The Structure and Functions of the Olivary Bodies. By W. Henry Kesteven.
11. On Spastic Paraplegia. By Samuel Gee, M.D.
12. A Case of great enlargement of the Pons, Crura Cerebris, and Medulla. By Percy Kidd.
13. A Case of Spasmodic Muscular Rigidity with Idiocy. By John Abercrombie, M.B.

1878.

14. On the Cervical Opisthotonus of Infants. By Samuel Gee, M.D., and Thomas Barlow, M.D.
15. A Case of Paraplegia with Sacral Decubitus. By Samuel West, M.B.
16. Cases of Disease of the Spinal Cord. By J. A. Ormerod, M.B.

1879.

17. A Case of Sclerosis of the Cerebral Cortex, with two other examples of Cerebral Disease in Children. By Norman Moore, M.D.
18. A Case of Cancerous Tumor of the Neck, simulating Myelitis. By J. A. Ormerod, M.B.
19. An unusual Cause of Hemiplegia. By W. E. Steavenson, M.B.

1880.

20. On some Intestinal Lesions in the Insane. By T. Claye Shaw, M.D.
21. Diseases of the Nervous System. By Samuel Gee, M.D.
22. Some Cases of Intra-cranial Syphilis. By J. A. Ormerod, M.B.
23. Case of Hystero-Epilepsy, with Hemianæsthesia and Chorea. By Walter H. Jessop.
24. Notes of Post-mortem Examinations of Cases of Hæmorrhage within the Cranium. By Norman Moore, M.D.

1881.

25. Diagnostic Symptoms of Tabes Dorsalis, with Cases. By J. A. Ormerod, M.B.
26. Tendon Reflexes in the later stage of Hemiplegia. By J. A. Ormerod, M.B.
27. Acute and Chronic Insanity. By T. Claye Shaw, M.D.
28. A Case of Pseudo-hypertrophic Muscular Paralysis in an Adult. By Percy Kidd, M.B.
29. Two Cases of Subacute Anterior Spinal Paralysis, with the Pathological Changes in the Spinal Cord of one of the Cases. By Charles A. Morton.
30. Two Cases of Cerebral Disease. By Samuel Gee, M.D.

1882.

31. A Case of Lesion of the Sympathetic Nerve in the Neck. By Samuel Gee, M.D., and John Abercrombie, M.D.
32. Two Cases of Hemiatrophia Facialis. By Walter Jessop, M.B., and Oswald Browne, M.B.
33. Clinical Cases from Dr. Gee's Wards. By Oswald Browne, M.B., and D'Arcy Power, M.B.

1883.

34. On Criminal Responsibility in the Insane. By T. Claye Shaw, M.D.

35. On a Peculiar Numbness and Paresis of the Hands. By J. A. Ormerod, M.D.
36. Five Cases of Tetanus, with some Remarks on its Pathology. By Anthony A. Bowlby.
37. On Trephining the Skull in Traumatic Epilepsy. By W. J. Walsham.
38. Cases of Ocular Motor Paralysis. By W. J. Collins, M.D.
39. On the Pathology and Treatment of some forms of Headache. By J. Lauder-Brunton, M.D., F.R.S.
40. On a Case of Amyotropic Lateral Sclerosis, with Microscopical Examination of the Nerve-Centres, &c. By R. J. Collyns.

1884.

41. Notes on the later Stages of Hemiplegia. By J. A. Ormerod, M.D.
42. On Degradation of Type in the Insane. By J. Claye Shaw, M.D.
43. A Case of Epilepsy, with Remarks. By Howard H. Tooth, M.B.

2. A table is given of 63 cases, which shows that laryngismus is more common in the first than in the last half of the year. In the first are recorded 58 cases, in the last only 5. The author attributes this to the dampness and chilliness of winter and spring. These conditions favour the diathesis and the paroxysm ; the former because the child is left indoors more than is good for it, and the latter because the damp and cold air may have an excitant action through the medium of the skin and lungs.

5. The term Phrenitis is chosen instead of Meningitis, because the inflammatory condition implied by the latter term was wanting. The cases related were 4, two boys and two girls, ages 1 year and 10 months, 4 years, 6 years and 5 months, 6 years and 6 months. The attack took place in very hot weather, but in one case not after exposure to the direct rays of the sun. The invasion followed the exposure within 24 hours. The symptoms were briefly as follows: Vomiting, diarrhœa, and shivering. High fever, 108·5 in one case, coldness of the extremities at the same time. There was marked delirium in three cases, sleeplessness, with picking at the bedclothes, and in the other, convulsions. The author regards delirium and convulsions as being of equal significance in children. Coma follows the delirium. In one case were symptoms pointing to affection of the base of the brain, *i.e.* irregular and infrequent pulse, unequal

pupils, right internal squint, deafness, cervical opisthotonus. In two cases the acute stage ended on the sixth day, but the patient has never completely recovered. One case became permanently deaf; the other went into a state of dementia, which, however, is improving. The other two cases died, one on the first and the other on the eighth day.

8. An account of 14 cases of epidemic cerebro-spinal meningitis. Five cases resulted in recovery. Six cases were under 18 years; the remaining eight were between 20 and 50.

11. Four cases are recorded, having as essential characteristics, constant rigidity of legs, or of legs and arms, which increases on handling the limbs, and disappears under chloroform. Other common characters are: that the lesion is congenital, or begins in early infancy. The ages of the children reported are respectively, 10, 8, 8, 3½ years. That of the four cases, two were males. That no tendency to heredity, neurotic or other, was discovered. The affected muscles act well to faradism, and show little or no atrophy, and no tremors. Intellect and sensation natural, no lesion of micturition. General nutrition good. Weakness of back in all cases. Choreiform movement of face in two. Tendency to convulsions in two cases. One suffered from painful cramps in the legs.

12. A girl, æt. 6½ years, in whom the most prominent symptoms were,—double internal squint, slight ptosis right, food lodged between teeth and cheek on the right side, impaired deglutition, deliberate speech, sight and hearing unaffected, but double optic neuritis. *Post-mortem*, the pons was found to be greatly enlarged, most on the right side; the swelling involved the posterior part of the crura cerebri and the left half of the medulla to the decussation. Right of the nerve compressed. Microscopically, was found great absence of nerve fibres in the tumour, which seemed to be due to a great general overgrowth of neuroglia.

13. A girl, æt. 3 years, quite idiotic, born healthy, symptoms began at 9 months old, and consists of spasmodic rigidity of one or more groups of muscles. The spasms may last a quarter of a minute, and then go off as rapidly as they came. On attempting to stand, the leg enters into a state of rigidity, with equino varus and tendency to cross. No sign of syphilis. Mother's father epileptic.

14. An analysis of 25 cases, 10 male and 15 female, illustrating a disease which affects infants under 2 years of age, and having for its essential sign a holding back of the head. The opisthotonus was not constant in all cases, but occasionally ceased for a time. Associated with it in some of the cases were,—rigidity of the limbs, epileptiform convulsions, hydrocephalus, the latter affection coming on some time after the opisthotonus was established. In 11 cases the disease was chronic, and termination unknown, but of the remaining 14, only 3 recovered. As to post-mortem appearances, no tubercles were found in the brain or membrane, but basic meningitis in all cases. In five cases the disease had existed from birth, two were certainly syphilitic, another probably so. Some of the cases resembled epidemic cerebro-spinal meningitis. There was no evidence of heredity, either ancestral or collateral. In three cases was a history of a fall before the onset of the disease, but fracture or laceration of brain or membrane was found after death. In two cases there was otitis interna.

17. These cases are related at considerable length and illustrated by woodcuts. The first was that of a child, aged 5 years. Her history was briefly, that after a fright she was affected by uncontrollable facial contortions on the left side, followed by left hemiplegia. Later, there supervened right hemiplegia with complete loss of speech. The right hemiplegia improved, but in about five weeks great contracture of the left limbs set in. There was slight hyperæmia of the optic discs. *Post-mortem* was found yellow softening of (1) the hinder part of the first frontal convolution; (2) the hinder part of the three frontal convolutions; (3) ascending parietal, and (4) ascending frontal convolutions; (5) callosa marginal convolution as far as the fissure of Rolando. This softening was symmetrical on both sides of the brain, except that it extended in the frontal region nearer to the olfactory sulcus on the right than on the left side. The vessels at the base were reported quite natural. The other cases described were,—one cerebro-spinal meningitis, the other of acute hydrocephalus, with accounts of autopsies in each.

21. Illustrated by the following cases: (1) Spastic paraplegia in infant and adult age. (2) Spastic diplegia. (3) Sclerosis of encephalon in inherited syphilis. (4) Paralysis due to compression of arm. (5) Traumatic myelitis, followed by muscular atrophy, (?) spastic rigidity. (6) Motor ataxia of legs, with paralysis of orbicularii oris. (7) Teichopsia. (8) Infantile spinal paralysis.

22. A careful record of three cases, among the symptoms of which are,—headache, more or less severe; drowsiness, eye symptoms, paresis and anæsthesia of certain limbs, epileptiform seizures. All three cases improved greatly under iodide of potassium and mercury.

25. An account of 21 cases of tabes dorsalis, arranged in three series: (1) Cases in which there was well-marked inco-ordination in the legs, causing difficulty in walking and inability to stand with the eyes shut. (2) Cases in which such inco-ordination was slightly marked. (3) Cases in which there was no inco-ordination. The relations of the inco-ordination to the pupil affections are also carefully discussed.

26. A suggestive paper showing the relation of the tendon-reflexes to rigidity in hemiplegia. The author finds that the knee-jerk in some cases of hemiplegia, even a long time after the attack, is the same on the affected as on the sound side. In such cases late rigidity rarely or never occurs. On the other hand, if the tendon-reflexes be increased on the affected side, rigidity may be predicted with comparative certainty. These points are well illustrated by tables comprising 50 cases of hemiplegia.

28. Man, aged 24; the calves had been noticed to be large for a very long time, but the more essential symptoms dated back about three years. A younger brother had been complaining of difficulty in running, but no enlargement of the legs had been observed.

Arms.—Triceps both enlarged. Biceps and flexors of forearm rather large. Pectorales and latissimi dorsi much wasted. Deltoids, infra- and supra-spinati, masseters, temporals, thumb-muscles and tongue, unaffected.

Legs.—Glutæi and thigh-muscles much wasted, except vasti externi. Calf-muscles much hypertrophied. Faradic irritability of hypertrophied and wasted muscles slightly diminished. Knee-jerk very feeble. This case is interesting from the fact that it is probably an example of Erb's recently described "Juvenile form of Progressive Muscular Atrophy."

30. (1.) *Hemichorea.*—A painter, æt. 54, no lead line or symptom of plumbism. Distinctly gouty. History of patient: Choreic movements of right hand five days before admission, soon spreading to leg. Never on left side. Died thirteen days after admission. In the left internal capsule, close to the thalamus at the junction of

its anterior and middle thirds, and separated from the lenticular nucleus by a band of healthy white matter, was a small patch of softened brain of the size of a lentil, and of an ochre hue. Also in the posterior half of the right thalamus was a small reddish cyst, the size of a millet-seed. The author emphasizes the following points: (1) That it was a case of simple chorea. (2) The onset was sudden, but there was no apoplectic symptoms. (3) No antecedent palsy. (4) No hemianæsthesia. (5) Cranial nerves unaffected. (6) The lesion was in the direct pyramidal tract. In cases of hemichorea associated with hemianæsthesia, the lesion has been found *behind* the pyramidal tract. (7) Probably embolic.

2. A second case of gelatiniform enlargement of the Pons Varolii.

31. Among the earliest symptoms was drooping of the right upper eyelid, closely followed by paralysis of the right side of the body. Right pupil half the diameter of the left, and does not dilate as fully with atropine as the left. Right forearm flabby, but with fair movement; can hardly move fingers at all. Faradic irritability of right superficial, and deep flexure of hand, pron. rad. teres, thenar and hypothenar, and interossei, abolished. Paralysis of both legs followed later, with absence of tendon reaction, superficial reflexes, and with complete anæsthesia. Patient perspired much about the head during sleep, and twice it was noticed that the perspiration was confined to left side of the head. Autopsy discovered a sarcoma connected with the first and third ribs, spreading into the spinal canal, and pressing on the cord. The right sympathetic was involved in the growth.

32. Two interesting cases, one of a girl, æt. 12, the other a woman, æt. 48. Illustrated by two photographs. In both, the atrophy made its appearance at an early age, and was associated with ocular troubles. In the second case it was preceded by intense pain in the side of the head. In both, the left side of the face was affected, and was marked by extreme atrophy of soft parts, with wrinkling of skin, loss of hair, and some wasting of the bones. In the child there was wasting of the muscles of the left arm, pectoralis major, deltoid and infra spinatus, with great depression of skin in palm, with wasting of middle and ring fingers. In both cases the muscles react well to faradism. Sensation is normal.

Mr. Jessop discusses the various theories of causation of this affection, and suggests that lesion of the Gasserian ganglion might account for the facts. The known connection of the sympathetic with the ganglion might have to do with the ocular affection.

33. These cases include, (1) a case of locomotor ataxia, associated with perforating ulcer of the foot; (2) a case of subacute anterior general and spinal paralysis; (3) a case of hereditary locomotor ataxia.

35. This article is illustrated by twelve cases, all women over 40 years of age except three. The symptoms always commence at night. The patient wakes up with numbness and formication, and even pain, in the hands or arms. There may be also some loss of power. Symptoms pass off after friction of the part, and much resemble that state induced by compression of the nerve-trunks. The affection, however, is often bilateral. The author compares it rather with a mild case of tetany, without the facial irritability. The use of bromide of potassium was attended with the best results.

36. In four cases of tetanus, Mr. Bowlby was able to make a careful post-mortem examination. In all, the nerves round about the wound were found to be perfectly healthy; the same may be said of the spinal cord, medulla, pons, corpora striata, and optic thalami, except in one case in which there was softening of the cerebrum just above the left Rolandic fissure, also in both corpora striata. In no case was the blood found to contain micro-organism.

The author summarises the current theories, and proceeds to show how baseless these theories are, both from pathological and clinical grounds. He himself inclines to the view that the disease is essentially one of blood-poisoning, in support of which he adduces these facts: (1) That the occasional idiopathic occurrence of the disease shows that local injuries are not essential for its production. (2) Its extreme prevalence in certain climates and localities. (3) Its preference for coloured races rather than white. (4) Its occasional appearance in epidemic form. The constitutional origin of the disease is shown strongly in the form called tetanus neonatorum, which attacks the children of coloured parents; the umbilical cord is always in an unhealthy condition, and bad hygienic surroundings always play some part in the affection. Here the peripheral nerve-theory falls to the ground, as there are no spinal nerves in the umbilical cord.

37. To this paper, which is principally of purely surgical interest,

is appended a series of tables comprising cases collected from all sources, European and American. They contain a record of 82 cases, in which most of the details are forthcoming, and 48 of which no details are recorded, making, in all, 130 cases, with a reference to each. In 74 cases complete recovery followed. In 18 cases improvement followed the operation. In 7 cases no improvement, and 30 cases died.

39. In enumerating the causes of headache, the author brings them under two heads: (1) Local irritation. (2) Causes having their origin in general conditions. Under the first head come decayed teeth; abnormalities of refraction in the eye; diseases of the ear, nose, throat, &c. Under the second head come indigestion, biliousness, constipation, fever, plethora, anæmia, rheumatism, gout, and albuminaria. He makes some interesting remarks on the position of the pain. Thus, in caries of the lower molar teeth, the headache is usually in the occipital region; decay of the upper molar causes temporal headache; while frontal or vertical headache may be associated with decay of the incisors and canines. The headache of anæmia is generally vertical. Frontal headache, with constipation, is relieved by salines; while that without constipation is best treated with acids or alkalis.

40. Full details are given of a case of a man aged 32, with no personal or family antecedents of any importance. The first symptoms consisted of wasting of the left-hand muscles, followed quickly by wasting of right hand and both arms, and at the same time dysphagia and other bulbar symptoms made their appearance. The paralysis, rigidity, and tremor proper to lateral sclerosis were of later occurrence. A full account of the post-mortem appearances, gross and minute, of the cord, nerves and muscles is given.

The author considers that the grey matter was primarily affected, the sclerosis spreading from this to the anterior columns and crossed pyramidal tracts.

41. Short descriptions of 20 cases of hemiplegia due to simple non-progressive cerebral lesion. The cases are arranged to elucidate the following points:—

(1.) Two cases to show that, contrary to the rule, the tendon phenomena are sometimes exaggerated without the supervention of contracture, and from cases in which they are more marked than on the sound side.

(2.) The tendon reaction is sometimes found exaggerated on both sides, and a case is quoted in which the paralysis is found on the same side as the lesion as well as on the opposite side. The author discusses the explanation of this post-hemiplegic paraplegia, and then describes,

(3.) Two cases of incontinence of urine after hemiplegia.

(4.) A case of hemiplegia, to which was superadded degeneration of the anterior horns of the grey matter, shown by rigidity, atrophy of thumb-muscles, and marked degenerative reaction.

(5.) Two cases of rigidity of hand and leg, alternated with involuntary slow movements (" mobile spasm "), which are easily provoked by any attempt to move the rigid limb.

(6.) Eight cases of post-hemiplegic epilepsy. In these the fits may occur at any age, and appear to have some direct relation to the hemiplegia, but do not make their appearance till some time after its occurrence.

Howard H. Tooth, M.B.

A Study of the Disturbances of Co-ordination in Children. By A. Erlenmeyer, M.D.—The case reported is that of a boy, the only child of related parents (first-cousins). He had the gait of locomotor ataxy, and restless movements of the arm. It is noted that he was seen by the late Professor Friedreich and Professor Erb : the former diagnosed spinal disease ; the latter, chorea minor.

History.—Well till the age of 5, then a fever of uncertain nature (? cerebro-spinal meningitis) ; five months afterwards the following nervous symptoms developed very rapidly—unsteadiness of gait, restless movements of arms, transitory tremulousness of the eyeballs. On admission (three years afterwards), there was well-marked tabetic gait, inability to stand with eyes shut, absence of patellar tendon-reflex : constant irregular movement of the arms, and (in connection with them ?) of the trunk and head ; movements of the facial muscles and right lower eyelid ; right internal squint ; right pupil larger than left ; both pupils acting normally. Speech described as " indefinite, difficult, showing irregularities in the mechanism of word-formations." No nystagmus. No affection of sensibility. The squint disappeared under treatment ; the speech improved ; but in other respects the boy remained in the same condition.

The author looks on the case as one of hereditary ataxia (locomotor and static), of which the fever was the determining cause.

J. A. Ormerod, M.D.

B R A I N.

JANUARY, 1887.

Original Articles.

ROTATION OF THE GREAT BRAIN.

BY ALEX. HILL, M.A., M.D.,

Fellow of Downing College, and Demonstrator of Anatomy in the University of Cambridge.

In the spring of 1885 I propounded, as part of a general scheme of the structure of the central nervous system, the theory, that the mammalian brain during its growth rotates upon itself.[1] The rotation produces a loop or kink, giving to the whole brain somewhat the form of a ram's horn, and bringing the part which was at first in front, on to the under side at the back. At its first formation the cerebral hemisphere of the mammalian brain is directed, like that of the reptile, straight forwards; the foramen of Monro opens into the back of its ventricle, the anterior end of the hemisphere bears the olfactory bulb. In the adult condition the foramen of Monro opens into the front of the ventricle, the olfactory apparatus is attached to the inner side of the temporo-sphenoidal lobe.

This view as to the alteration in position of the great brain was the almost necessary deduction from certain conclusions to which I had come as to the connections of the olfactory tract, for I believe that I have been able to prove a direct continuity between the olfactory tract and the anterior end of the optic thalamus, *viâ*—the fimbria, fornix, descending pillar of the fornix, corpus mammillare, and bundle of Vicq d'Azyr. That the olfactory nerve is connected with the anterior end of the optic thalamus I have many reasons for believing, and the circuitous route which I have sketched out is not by any means difficult to follow. Its path is easily broken off from a hardened brain, as shown in the accompanying sketch, and seems to form an organically distinct part of the brain; and besides we have, as I shall show presently, a certain amount of embryological evidence in favour of this extensive displacement of the olfactory tract. The path here sketched out for the fibres of the olfactory tract almost necessarily presupposes a rotation of the brain; but it is not this connection only which the theory renders intelligible. Many other features in the plan of formation of the great brain are accounted for by this supposition of its rotation. Some of this corroborative evidence I set forth in the thesis already quoted, and the object of this paper is to point out certain other peculiarities in the structure of the brain which have recently attracted my attention, as being still more favourable to the theory.

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[1] 'The Plan of the Central Nervous System.' Cambridge: Deighton Bell & Co., 1885.

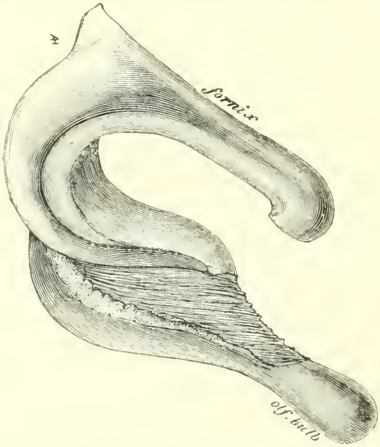

Fig. 1.—Fornix, pyriform lobe and olfactory bulb, broken off from hardened brain of Ox.

Among *external appearances*, the most suggestive is the progressive closing in of the fossa of Sylvius. The fissure of Sylvius, as is well known, is not for the greater part of its extent in any way comparable with the other fissures of the brain. Instead of being formed like other fissures, as a narrow slit-like depression on the smooth rounded surface of the brain, it makes its appearance before any fissure, if we except the so-called rhinal fissure, as a dimpling of the mid part of the outer surface which gradually deepens into an extensive shallow fossa. The appearance of this fossa, is due, as I believe, to the traction exercised upon a part which will subsequently become the temporo-sphenoidal lobe by its attachment through the olfactory tract and bulb, to the cribriform plate of the ethmoid. As the great brain swells, its olfactory part being attached to the skull, a pitting of the outer surface is the necessary result. The fossa of Sylvius is subsequently converted into the fissure of Sylvius, a Y-shaped fissure, with a common portion leading into an anterior and a posterior limb by the outgrowth of the surface of the brain which borders it in three portions, the operculum coming down between the limbs of the Y; the frontal and temporo-sphenoidal lobes closing up beneath the operculum. The want of homology between the fissure of Sylvius and other fissures is shown, by its enclosing a considerable cortical area, the Island of Reil, which formerly formed the floor of the fossa. The radiating fissures of the Island of Reil indicate plainly enough the direction in which the traction was exerted.

When viewing the outer surface of any mammalian brain, it is easy to convince oneself that it has undergone a rotation, and that the position of its parts has been altered in such a way from the form maintained amongst fishes, amphibia, and reptiles (for special reasons it is better to exclude from the comparison the bird's brain), that the temporo-sphenoidal lobe of the mammal's brain corresponds to the anterior end of the brain of lower vertebrates.

Following closely upon the external form, the delimitation of the cortex into areas associated with peripheral nerves, presents itself as a test by which to try the theory. Recently, principally as the result of ablation experiments, the function of almost every part of the cortex has been determined, and although we are as yet far from possessing an accurate industrial map, we can nevertheless speak with assurance as to the general distribution of work among its various territories. Undoubtedly the anterior end of the brain, particularly the Rolandic area, is in connection through the mediation of grey matter of the spinal cord with the body nerves. The localities in which sensations are received through the 8th and 5th nerves, lie somewhere in the neighbourhood of the fissure of Sylvius; the 2nd nerve has its area of cortical distribution in the occipital lobe—the 1st nerve on the inner side of the temporo-sphenoidal lobe. I have attempted to show elsewhere[1] that the observation of brains of animals remarkable either for preponderance or for deficiency of particular senses, will help us better than even direct experiment to map out the cortex into functional areas. Whatever method be adopted for obtaining the necessary data, any sketch of the brain in which functional distribution is indicated, shows clearly enough that the order from before backwards in which the areas connected with the several nerves are situated, is the reverse of that which obtains among the nerves themselves. For embryological reasons, which will be presently stated, it is only necessary to treat of the larger groups of cortical centres. The arrangement of their sub-centres, as determined, for instance, in the marginal gyrus by Horsley and Schäfer, may for the present be neglected. The body nerves have their cortical areas in front, and are followed by the nerves of the head; of these latter the optic nerve is indirectly connected with the back part of the upper surface, while the olfactory has its area in what I take to be the reflected portion. I imagine that the rotation over from before backwards is accompanied by a certain rotation from above outwards.

Passing now to the *internal structure* of the hemisphere, so far as the course of fibres within its white substance is sufficiently well known to help us, we find further evidence of rotation. Leaving out of consideration all doubtful tracts, we have the classical observations of Gratiolet, which have been frequently confirmed, to show that the anterior end of the optic thalamus is connected through the "inferior stalk" with the sphenoidal lobe, the posterior end of the thalamus through the "optic radiations" with the occipital lobe. Numberless recent observations have placed the connection of the grey matter of the spinal cord with the frontal and anterior parietal lobes by means of fibres occupying the anterior part of the internal capsule beyond doubt. These three sets of fibres, anterior part of internal capsule, optic radiations, and inferior stalk, obviously cross one another, as shown in the accompanying diagram (Fig. 2), plainly indicating that the cortex of the cerebrum and the grey matter of the basal part of the system extend in contrary directions. I am afraid, however, that I can hardly render intelligible the force with which the crossing of these three sets of fibres appeals to myself, without touching upon my view as to the fundamental plan of construction of the central nervous system. The system arises as an involuted tube of epiblast. Throughout the spinal region the inner wall of the tube becomes grey matter; the outer wall constitutes the white columns of fibres. In the cephalic region a second layer of grey matter, the cortex, is added on the outer side of the tube; all the grey matter of the system grows from one or other of these grey tubes. In the spinal cord, which only contains the central tube of grey matter bordering the central canal, are situated all the primary centres of the body nerves; indeed, the grey matter is entirely

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[1] "Hunterian Lectures, 1885," reported in 'Brit. Med. Journal,' March 14, 21. Report 'British Association,' 1885, p. 1054.

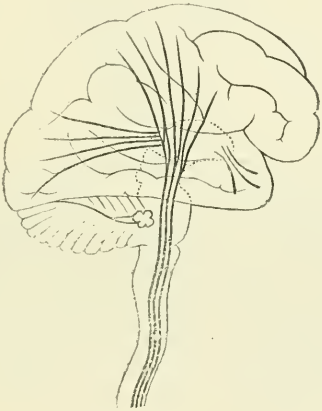

FIG. 2.

used up in forming nerve-cells and plexus for the reception of sensory, and the origin of motor fibres. Travelling up into the brain, no break in the continuity of the grey matter is to be observed. It lies in the floor of the fourth ventricle, around the iter, and in the sides and floor of the third ventricle. No one doubts the continuity of plan as far as the anterior end of the iter; but hitherto it has not been recognised that the grey matter bordering the third ventricle is in functional continuity with the grey matter of the cord. The optic thalami are treated of as "basal ganglia." The olfactory and optic nerves are deprived of primary centres such as are accorded to all other sensory nerves. It is impossible for me here to point out the modifications in structure of the central system, due to the situation peripherally in the olfactory bulb and retina of some of the grey matter, which elsewhere enters into its constitution. An explanation of this will be found in the reports of my Hunterian Lectures of last year.[1] In this paper I must be content to state merely, that I regard the optic thalami as constituting the anterior extremity of the central grey tube or basal part of the central nervous system: if this position were conceded, the attachment of the extreme anterior end of the thalami with the temporo-sphenoidal lobe, of the back of the thalami with the occipital lobe, and of the spinal cord with the front of the hemisphere, would supply the strongest possible evidence of rotation.

That degeneration of a portion of the internal capsule, of the pyramid, and of the pyramidal tracts of the opposite side of the spinal cord follows extirpation of the "motor area" of the cortex, is universally recognised. The "motor area" is a congeries of local centres. The fibre-bundles which lead from local centres in the cortex to the primary motor centres in the cord, must occupy, one would suppose, discrete regions in the internal capsule and pyramidal tract. As yet considerable mystery involves their course, for it is not certain that only extirpation of the centres for the limbs and trunk induces descending degeneration of the cord. In some cases degeneration of the cord seems to have followed injury to the centres for head muscles only. It has also been observed that injury to the centre for the arm may be followed by a degeneration descending lower in the cord than the primary centres for the muscles of this limb. It is very interesting to notice, however, that the only observers[1] who have turned their attention to this matter have recorded their opinion, that the anterior part of the motor area is connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the anterior (ventral) part of the lateral tract.

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[1] 'Brit. Med. Journ.,' March 6, 13, and 20, 1886.

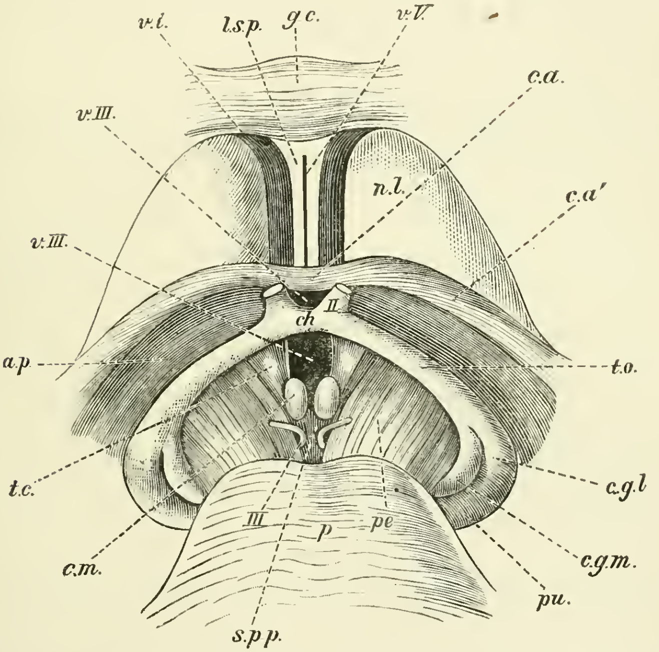

FIG. 3.—*l.s.p.* Septum pellucidum. *v.l.* Anterior horn of lateral ventricle. *n.l.* Nucleus lenticularis. *c.a.* Anterior commissure. *v.III.* Third ventricle. *g.c.* Genu corporis callosi. *c.g.m.* Corpus geniculatum mediale. *c.g.l.* Corpus geniculatum laterale. *c.m.* Corpus mammillare.

Passing now to the consideration of another intra-cerebral tract, we find in the arrangement of the fibres of the anterior commissure evidence of rotation of a most convincing kind. The accompanying illustration, which is a copy of the picture on page 490 of Schwalbe's 'Neurologie,' accurately represents the peculiar twisting which the anterior commissure has undergone. It looks as if the middle of the commissure had been fixed, while its ends were twisted in such a way that the fibres which cross the median line on its under side, pass round in front to reach the upper side at its extremities. Just such a torsion, it is to be noticed, as would be produced by the rotation of the hemisphere, which I imagine has taken place, for it so happens that the rounded anterior commissure, lying just below and in front of the foramen of Monro, is exactly in the axis of rotation.

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[1] Langley and Sherrington, 'Journal of Physiology,' v. p. 49; and Langley, "Critical Digest of Recent Observations on Degeneration, and on Nerve Tracts in the Spinal Cord." 'BRAIN,' April 1886, p. 109.

In addition to the demonstration which the twisting of the anterior commissure affords of the fact of rotation, peculiar interest attaches to it, since it enables us to fix approximately the time at which rotation takes place. It must occur after the commissure is formed. There can be no doubt that the anterior commissure appears very early in the mammalian brain, for although we have, as far as I know, no exact observations as to the time of its appearance in the individual, this much may be inferred from its great prominence in the lower forms of vertebrates. It is *par excellence* the commissure of birds and reptiles. The existence of a rudimentary corpus callosum in birds was pointed out by Meckel,[1] but the view of Stieda,[2] that no such structure exists, is endorsed by such recent observers as Rabl-Rückhard[3] and Bellonci.[4] On the other hand, Stieda recognises the existence of a corpus callosum in reptiles, but denies it in amphibia, where Leuret, Blattmann, and Reissner[5] had found it. In fishes, the commissures appear to be fused, so that no distinction between corpus callosum and anterior commissure is possible. Quite recently Osborn[6] has shown that both commissures may be found in reptiles, birds, and amphibia, although in the two former classes the corpus callosum is very ill-developed. A good deal of the confusion in this matter has arisen, as it appears to me, from a misapprehension of the general homologies of the brain in the sub-mammalian orders. The cerebral hemisphere of the bird, and to a less extent of the reptile, is homologous, not with the entire hemisphere of the mammal, but, as pointed out by Cuvier, with little more than the corpus striatum. In the bird the cerebral cortex is very rudimentary, and consequently the corpus callosum, which is the commissure of the supra-ventricular part of the hemisphere, is ill-developed also. The anterior commissure belongs to the infra-ventricular portion of the great brain. Some confusion is due also to the complexity of the anterior commissure, which consists in mammals of two portions; (first) the pars olfactoria, which is the commissure of the inferior frontal regions (and ? the nuclei caudati), and (second) the pars temporalis. The internal olfactory tract also takes advantage of the situation of the anterior commissure to cross over to the opposite side. In sub-mammalian orders, judging from Osborn's description of Rana and other forms, the olfactory tracts reach the grey matter on the opposite side of the third ventricle independently of the commissure, which still consists of two parts, to which Osborn applies the mammalian names olfactoria and temporalis. It is open to consideration whether the homologies are not exactly reversed. For the purpose of this paper, however, we may leave out of consideration the difficulties in tracing the phylogenetic history of the anterior commissure, and content ourselves with the assurance that this commissure, in harmony with its ancestral history, is certain to develop early, and we may infer from its twisting that the mammalian brain had already reached a reptilian stage in its development at the time that rotation occurred.

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[1] Meckel, 'Deutsches Archiv für die Physiologie,' Bd. 11, 1816.

[2] Stieda, 'Zeitschrift für Wissensch. Zoologie,' Bd. 20, 1870; also Bd. 25, 1875.

[3] Rabl-Rückhard, 'Zeitsch. f. Wiss. Zool.,' Bd. 30, 1878.

[4] Bellonci, 'R. Acad. dei Lincei,' Memorie xiii., 1882.

[5] Reissner, 'Bau des Centralnervensystems der ungeschwänzten Batrachier,' Dorpat, 1864.

[6] Prof. H. F. Osborn, 'Morphol. Jahrbuch,' 1886, p. 223.

The *minute structure* of the brain also affords some evidence of rotation, for the margin of the cortex mantle is for a varying distance, where it skirts the under side of the crus cerebri, invested in a sheath of grey matter quite different in structure from the cortex itself. This sheath of small-celled matter, the fascia dentata, which forms part of the cornu Ammonis, receives the thin edge of the cortex mantle. It commences beneath the uncus at the apex of the sphenoidal lobe, and extends along the course of the posterior pillar of the fornix, reaching in some animals (particularly rodents) almost to the point where the fornix dips down in the anterior pillar. The fascia dentata is, in my opinion, identical in constitution with, and indeed a continuation backwards of, the nuclear (glomerular) layer of the olfactory bulb. In straight-brained animals the hemisphere terminates in the olfactory bulb. In twisted brains the olfactory bulb is attached to the apex of the pyriform lobe (the gyrus uncinatus of human anatomy). The continuation of its nuclear formation, as the fascia dentata, obliquely beneath, behind, and above the crus, simply shows the direction in which rotation of the great brain has taken place.

Ontogeny.—Evidence obtained from direct observation of the brain in successive stages of its growth must obviously, when the subject is thoroughly worked out, be the first to be submitted in proof of the rotation of the hemisphere. On account, however, of its scanty nature as yet, I have reserved it until the end of this paper. Partly its deficiency is due to the absence of research, partly it depends upon the peculiar nature of the organ itself. Unlike other tissues of the body, the nervous tissue is peculiarly plastic. In the course of its growth it may receive marks of torsion and other displacements, but while in other tissues such marks permanently remain, in the brain they are obliterated by further growth. The manner in which its mass is added to also has an important bearing upon its form. It must be remembered that only the cellular elements enter into its original constitution; the fibres are but processes of the cells. Of the primitive epiblastic cells involuted to form the nervous tube, some, in all probability, are favoured above the rest and become the functional nervous elements, while to the others only subsidiary, supporting, nutritive and insulating functions are assigned; they become the neuroglia and myelin cells; just as in the case of each little group of epithelial cells involuted into a follicle of the ovary, one only becomes an ovum, the others minister to its wants. Most likely, therefore, the lines along which nerve fibres are to grow are in many cases laid down, before the fibre itself appears, by the chain of accessory cells which have taken up their position along its route, but it is open to us to suppose that, in other cases, the route chosen by a fibre depends upon convenience at the time of its appearance. If, owing to a displacement of the cortex, it can reach its destination by a shorter route than the tract occupied by neighbouring fibres, which being fully developed at the time of the rotation of the brain, shared in the displacement of the cortex, it is open to it to leave the original tract, and thus the primitive arrangement of the constituent parts of the brain may be masked. Despite, however, the obliteration of the indications of rotation which these two causes are likely to produce, we still can see, I think, in the changes of form which the great brain undergoes signs sufficient to prove that rotation does occur. Undoubtedly this is the conclusion one is likely to draw from the examination of three such pictures as are here inserted:

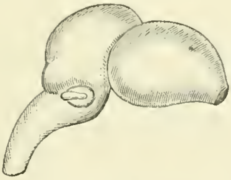

Fig. 4.

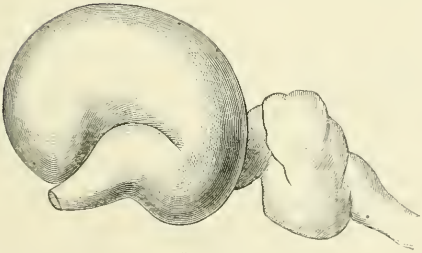

Fig. 5.

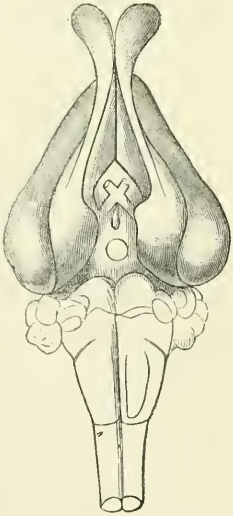

Fig. 6.—Ventral surface of brain of fœtal rabbit, after Löwe.

In Fig. 4, copied from Löwe,[1] a very early stage in the growth of the brain is shown, the hemisphere is directed forwards and bears the olfactory bulb at its anterior end.

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[1] Löwe, 'Entwickelungsgeschichte des Nervensystems,' Pl. I., figs. 1 and 2.

Fig. 5 represents the appearance presented by almost any mammalian brain after the formation of the rhinal fissure. The olfactory bulb is now attached to the extremity of the pyriform lobe, and the suggestion of a folding over of the whole hemisphere is very strong. The pitting of the side of the brain due to the tension on its posterior end produced by the attachment of the olfactory bulb to the cribriform plate, is beginning to appear as the fossa of Sylvius. Fig. 6, again, after Löwe, is inserted for the purpose of showing the continuity of the olfactory bulb, pyriform lobe, and gyrus uncinatus. It is curious to notice how the backward growth of the hemisphere increases the size of the loop. In the brain of a sheep, for instance, the fimbria (posterior pillar of the fornix) is transverse at its first appearance. In marsupial brains it never becomes more than very slightly oblique. The higher the animal in the mammalian scale, the more nearly does its fornix system assume a longitudinal direction.

Of the earliest stages of the growth of the brain we have very few observations, but Marshall's[1] discovery that the olfactory nerve arises, like all other sensory nerves, from the neural ridge, and that it is then pushed down in front by the outgrowth of the cerebral hemispheres, to be again caught up in the cortex mantle, opens up great possibilities with regard to the relation of the olfactory apparatus to the hemisphere, and prepares us, although the research has gone no further, to find an arrangement in the first nerve quite unlike that of the others.

Phylogeny.—The reference already made to the marsupial brain has drawn attention to the fact, that in the lower mammals the rotation has gone less far than it has in the higher members of the class. Whether or not it is a phase in the development of any sub-mammalian animals, I am not prepared to state. Certain it is that, if we descend as far as the reptiles, rotation is out of the question. As already pointed out at the commencement of this paper, the reptilian brain, like the brain of the early mammalian embryo, is directed forwards, its ventricle communicating with the third ventricle behind through the foramen of Monro, and ending in the

[1] Marshall, 'Quarterly Journal of Microscopical Science,' July 1879.

ventricle of the olfactory bulb in front. The great gap between the bird's and mammal's brains, although bridged over by the brain of the monotreme, renders it unlikely that the link between the straight and the rotated brain will ever be found. The peculiar deficiency of cortex in the bird's brain renders rotation unnecessary, for I do not think that the *reason for rotation* is far to seek. If we compare such an extreme type as the crocodile with any mammal, we notice a contrast in the whole architecture of the head. The reptilian head is directed forwards; is elongated in the direction of the axis of the body. The mammalian head approaches a globular form. With the greatly increased size of the brain as we ascend in the vertebrate scale, economy of space for its reception becomes necessary. Doubtless, for other reasons also, a shortening of the head is desirable, and the larger brain can only be packed inside the shorter skull by throwing its hemispheres over into loops.

SOME OF THE RELATIONSHIPS BETWEEN EPILEPSY AND INSANITY.

(Read before the Neurological Society of London.)

BY C. H. SAVAGE, M.D., F.R.C.P.

THOUGH I have no great practical experience with epileptics as a class, yet, having so much to do with the insane and with their relations, I have had opportunities of seeing at least some of the relationships which exist between the two classes, and therefore, in deference to the wishes of Dr. Hughlings-Jackson, I shall give you the result of this experience. I shall not give any definition of epilepsy, for I feel that at present we have to look upon it as a "disease" provisionally, and that the time is at hand when it will be looked upon only as a group of symptoms which happen to occur together, and which will be seen to occur together under many varying conditions.

I would only say, for convenience, that I make clinically two main divisions among epileptics, namely, the neurotic and the accidental, the functional and the organic, if I may use these terms without connoting too much. I mean to imply, that there are many cases in which the epilepsy seems to me to belong to a state of inherited nervous instability; but beside these, there are many other cases in which, sooner or later, we shall learn that there are definite lesions in the brain itself, giving rise to the epileptic symptoms. The first set of patients belong to the insane class; the second, on the other hand, need have no such connection, but are allied to those cases which follow injury or coarse brain changes.

But returning to the consideration of the relationships between the two disorders—and by relations I wish to imply not only likeness but contrast. Epilepsy and insanity are alike in origin. Similar causes will produce insanity and epilepsy. Probably both are often the result of one cause, and we must never forget this in looking at the relationships between these disordered states.

As insanity is very often the direct product of insane parentage, so epilepsy may result from epileptic parentage. But further, there is a family peculiarity which we call neurotic—from neurotic parentage we often see both insane and epileptic offspring. By neurotic parentage I mean that there are certain degenerate families in which we meet great nervous instability, a tendency to show bodily weakness by nervous perturbation. This nervous instability may be shown either in loss of control or loss of power, and this loss may affect either the motor or the mental side chiefly.

But to further develop this fact, that epilepsy and insanity are allied by origin, I must give some facts as examples.

We often meet with both insanity and epilepsy in the same family; we may meet with epilepsy in a parent and insanity in the children, or we may meet with insanity in the parent and epilepsy in the children.

In my experience it is very common to see the epilepsy in the collateral branches of the insane family, but we must not forget the important fact, that there are more members of the collateral branches than of the main stem.

Besides neurotic inheritance, I believe that there are some special causes of insanity which are more likely to produce epilepsy in the offspring. Thus I believe that insanity produced by alcohol, or by coarse brain injury, is very likely to re-appear as epilepsy in the next generation. I do not give this as a dogmatic faith. But I have seen it occur myself, and I find that writers on alcoholic-insanity give many cases in which this connection is manifested.

I think there is a difference then between the children of certain insane persons and those of certain others, suffering from insanity, and I would put it thus; that whereas it is common to meet with the children of the truly neurotic parent suffering from idiocy, moral insanity or moral obliquity, or from the more chronic and organised forms of insanity with hallucinations, such as those cases described as delirium of persecution or paranoia, it is more common to meet with epilepsy in the offspring of those who have become insane from some local degeneration or disease of the brain. I trust this subject will be studied and supported, or refuted, according to the truth that is in it.

Epilepsy may occur then in the same family as insanity, being produced by similar predisposing and exciting causes. And there are some other general conditions which set up both the one and the other in families which have been free from any taint of neurosis, and this too is a fine field for the investigator. I subjoin some instances.

The eldest daughter of an eccentric but highly gifted man died of acute delirious mania, which followed an attack of measles; another daughter suffered from chronic insanity, with delusions of a very fixed nature; while the son suffered for some years from epilepsy.

In one other a still more interesting relationship existed, for of twin children, one suffered from insanity and the other from epilepsy.

In other cases, idiocy and insanity, and epilepsy and idiocy, have presented themselves in different members of the same family.

So much then for the relationships of origin, and I need not repeat what I have so briefly put before you.

I now pass from the development of epilepsy in the same family to the occurrence of both epilepsy and insanity in the same individual.

Epilepsy may be preceded by some mental disorder, and this disorder may vary in degree from the simplest hallucination of a sense, occurring as an aura, to a furious outburst of complex but uncontrolled actions. Hallucinations of any of the senses may occur, and though I have frequently met with hallucinations of smell, I would not like to say more, than that they occur in my experience more frequently with epileptics than with those suffering from insanity. We meet with hallucinations of sight, and these may assume the character of an explosion, if I may use that term; they may appear as flashes or sudden rushings of light; the hallucinations of sound, too, may be explosions or "voices," and though I do not attribute much importance to the experience, I think that what appear to be directing voices do occur sometimes before as well as after the fit. The hallucinations preceding the fit may colour the acts performed after the motor disturbance has passed off, and a threatening voice as an aura may be the cause of a violent attack, which is performed during the post-epileptic automatic state.

As a rule, I believe these auras or these delusions are, like the rest of the fit, regular and constant in their origin and their course. It is quite impossible to separate the periods clearly, for if very slight fits occur, the actions which appear to replace the fit really follow it.

In the following case there seems no doubt the symptoms—mental—are mixed up with the fit. The case is known to your chairman—Dr. Wilks.

A busy provincial lawyer, especially when over-anxious, as at election times, had what he called a very strange experience. Tired, he would go to bed and sleep fairly well, but he would have a very painful dream. He would feel that he was brushing his teeth rather violently, and that suddenly the tooth-brush would slip from his hands, and then that he was choking. He would struggle for his breath, and during this struggle he would lose himself, and only remembered the dream in the morning, when he woke, dull and tired, to find a spot of blood on his pillow. As I said above, this dream repeated itself in every particular, like other true epileptic seizures.

It seems to me that the step between this man and the somnambulist is very short; the unconsciousness of the latter makes the difference. I cannot help thinking that a very little more stimulus would have made the lawyer do something, in his struggle for breath. Sense perversions, or more organised insanity, then, may precede epileptic fits.

In certain cases insanity is supposed to take the place of the fits altogether, and it is not necessary for me to go into all the evidence which has been brought forward to support the existence of "L'épilepsie larvée;" my own opinion, supported by Dr. Hughlings-Jackson and others, is that in most of these cases a fit of some sort, great or small, does occur before the strange acts. This marked epilepsy is less common in England, or at least has been less commonly studied in England, than in France, and it is interesting that France should produce such peculiar varieties of neuroses; there seems to me to be a specially nervous element for good and ill in that nation. I have met with but few cases of true masked epilepsy, and in none have I been convinced that no fit had occurred. The acts similar to those described by French authors are not so very rare, and I would give a case which I look upon as a good example of the kind of fit which is supposed to be masked.

A young labouring man, belonging to a nervous family, that is, to a family where neuralgia and mental peculiarity occurred, took to drink, developed epilepsy, and later became insane. From the epilepsy developed violent outbreaks of mania, and for these he was sent to one of the county asylums. After treatment, he recovered from his insanity, and had almost got rid of his fits, these only coming on at distant intervals. As they were always preceded by a distinct aura, he was able to manage himself very well; he continued at work and was steady. There were, however, occurrences for which he could not quite account, and there were lapses in his life he could not fill.

The mystery was cleared up in this way. As he was walking along a country lane he felt his fit coming, and loosened his collar and necktie, as he had learned to do in the asylum. He got to the roadside and lay down; on recovering his senses he could not understand the nature of things, for he found himself at the distance of some miles from where he loosened his collar. He heard later that he had been seen and met by some who knew him, and that he passed them without appearing to recognise them. His gait, too, was said to have been staggering, so that his neighbours not unnaturally thought that once more he had taken to drink. Doubtless the history was this, that after the aura came the fit; then there followed the slight rest, followed by an automatic state, in which the journey was pursued, till a period of exhaustion set in, associated with sleep, and the awakening took place at a distance from the site of the fit.

Later I shall show how this case connects the epilepsy with epileptic fury.

Epilepsy may not only be preceded and replaced by insanity, but very commonly indeed it is followed by mental disorder. We shall in this great class find hundreds of patients who fill the asylums for chronic lunatics.

Epilepsy seems to have a very destructive influence on the mind, and here it will be well to study some points in relationship to the mental weakness following epilepsy.

Is the weakness the result of the exhaustion produced by the severity of the epileptic discharge, or is the destruction due to mental instability (inability to construct and repair), resulting from the disturbance due to fits; or do these epileptic attacks set up a process which does not end with the fit?

There is one other consideration which I think deserves more careful study. It is often said we have too many cases described as epileptic, as though there were only one set of mental relations to one set of epileptic ones.

I believe that study will show, that among the hundreds of epileptic lunatics there are many sub-groups quite worthy of names. There are certain cases in which the convulsions are associated with certain sets of mental perversions, and there are certain other cases in which the fits take other relationships to the mental disorder. This field is a large one, and one at present but badly tilled.

I am constantly talking of "functional" disturbance; and though I know this is rather heterodox, when we are supposed to find a physical basis for everything which occurs, yet I for one am forced to see cases in which I cannot help blaming the function before I do the organ. And in studying epilepsy I wish to say that I believe there may be functional equivalents to general paralysis of the insane, and just as we have decay of power associated with loss of control, just as we so commonly have fits towards the end of general paralysis, so I believe among the chronic epileptics of the asylums will be found patients who are perverted functionally, and with recurrent discharges of an epileptic nature. This part of my paper is highly theoretical, and is very open to debate. I merely suggest the possibility, and I shall be glad to hear more from others.

There is no doubt that insanity of the weak-minded type follows the more *frequent* rather than the severe fits. I should like to hear whether it is true of epilepsy as of insanity, that those who are epileptic by origin can bear much more nervous disturbance than can those who are apparently of good stock. The more unstable are more easily upset, but again more easily reset. It is true that, in some cases to be referred to again, a single fit is enough to upset the mind and render it weak for life, but this is not the rule; the constant repetition of fits is supposed, by some, to be like constant spirit-drinking, a cause of constantly recurring hyperæmia which tends to degenerative changes.

It is self-evident that constantly recurring disturbance of function will lead to the injury and destruction of the organ, and I do not believe, notwithstanding all that has been said about the special circulation in the brain, that disturbance of the motor areas constantly recurring, will leave the rest of the brain unaffected by the neighbouring change.

The fact remains that there are a very large number of chronic epileptics who are weak-minded. Also, I believe the study of these cases will be as useful to the physiologist and to the philosopher, as is the study of the weathering of the rocks to the geologist.

There are almost certainly among these weak-minded epileptics at least several groups in which the epilepsy is but a small part of the disordered process.

We have to learn that all fits are not alike, or always associated with a special sort of mental disorder, and, what is more, I shall have to point out that fits may not always be the worst part of the diseased action. The relation of the fits to the age of the patient is important. We all recognise that constantly recurring fits produce weakness in mind, and we also see that the younger the age when the fits appear, the worse is the prognosis, so that fits in infancy often prevent all further mental development.

Disturbed nervous conditions and epilepsy are not uncommonly associated with adolescence, and here we have to do with a more stable age than childhood, yet one eminently unfit for severe shocks, and likely to suffer greatly in mind by fits. As manhood advances, the onset of fits has a graver meaning, but then only as probably being associated with other and more tangible changes in the brain itself. And when age comes on, the same thing holds of age as of childhood, the power of the fit for evil seems very great; in the one case, the edifice is not yet built and founded firmly; and, in the other, it is tottering to its fall.

So far, I have inverted the usual order by beginning with the study of the more chronic mental changes produced by epileptic fits, before examining the more acute symptoms which may follow directly on the fit. I have now something to say about the acute mania which frequently follows an epileptic fit.

The fit may be severe or not; the patient may sleep for a longer or shorter time, and may suddenly or gradually become excited. He may perform the most complex actions, and may speak and appear to hear what is said to him, and yet in most of these cases he will have no recollection on his recovery of anything which he has done. As I said before, the state is a condition allied to that which is called automatic, and his acts are in many cases a direct result of some sensory or higher intellectual discharge. If I may use the term, the machine seems to be set in motion by some sensory or other disturbance, and works along the usual track as if it were a mere calculating machine. I know in using the term "discharge" I shall lay myself open to criticism, on the ground that there cannot be any, or at least many, intellectual centres answering in any way to motor centres; but I can best explain what I mean, by referring to other conditions in which, the higher control being removed, there is a similar discharge, that is, an unrestrained running away of force along certain lines.

Thus, on the one hand, we meet with monkey-like idiots who are never still for a moment; they are never saving or storing any force for future use; the force seems to be expended in any reflex way as soon as it is made, and we see a very similar state of things in the earlier stages of dementia in some elderly persons.

The higher control removed in both these cases is seen in the unrestrained exhibition of energy, and, as a rule, it is mischievous and destructive, and with but little memory of what had occurred. I do not wish to push the likeness too far, but I only use the comparison to explain what I mean by intellectual discharge.

Epilepsy may be followed at once by fury, but, as a rule, there is a period of rest between the fit and the fury.

The mental disorder may be senseless destructive violence without any apparent aim or object in view, or it may have the aspect of design. In this latter case it may depend on the delusion or the sensory perversion which started with the aura, or it may result as a kind of intellectual reflex due to some sense impression arising during the fit or immediately after it. The actions may point to some fear of being followed or persecuted, and may be perfectly consistent with ideas of self-defence. There is nothing in any way irrational in the acts themselves in many cases, though in others these acts are incoherent, if I may use such a term, that is, they are "mixed," part of one set of actions being confused with another set. He would be very rash who would pretend to dogmatise and qualify the nature of what acts may or may not be done in an unconscious epileptic state.

The duration of the maniacal excitement is another point of great interest to me, for it seems as if there may be a mental side to the status epilepticus, just as there is a motor one. In post-epileptic mental disorder it seems as if a process were often started which is very hard to reverse or stop.

The starting of epileptic mania by a sudden muscular disturbance recalls to my mind those cases of insanity in which some acute cause—such as delirium due to fever, alcohol, belladonna, or even to chloroform—has sufficed to start a maniacal process, which has continued for weeks or months. One other point of interest is that there is a likeness between the two diseases, epilepsy and insanity, from the treatment, bromide of potash having as high a reputation for relieving the one as the other. I do not for a moment wish to disparage the use of bromide in both disorders, but it would be unreasonable to suppose that a disorder, which I believe to be but part of many morbid processes, and arising in many varying conditions, is to be treated always by one remedy because one of the chief symptoms happens to be fits.

I hear from some asylum superintendents that they do not give bromide at all now, as they find that the patients do not seem to do so well when these fits are entirely suppressed; and I should, from my point of view, say that that asylum is not the best where the patients are the most quiet, if the quiet be the result of drugs. That I have ground for believing that harm may follow bromide, I will show by giving one example from many.

A girl of weak intellect from childhood developed fits, occurring at long intervals, mostly at night; these never attracting much notice till she approached maturity, then they were vigorously treated, and the child became much worse in mind, so that she was now almost always restless, excitable, destructive, and dirty. The mother's own words were, "Doctor, there are worse things than fits." She then told me that when the child had the fits she was always brighter and more companionable for weeks after, and only unruly just before their onset. This case is not singular. I could record several others, but one suffices for my object.

Another relation calls for attention.

Either epilepsy or insanity may be replaced by other neuroses. I know that epilepsy may be replaced by migraine or asthmatic attacks, and I have seen several good examples myself in which insanity has been replaced by similar disorders.

I have incidentally said that there are worse things than epilepsy, and I should not feel that I had completed my paper without referring to cases in which epilepsy seemed really to have had a chief part in restoring the mental balance. First of all, my attention was called by several cases of rather chronic insanity, in which severe fits were followed by recovery. In one, a case of profound melancholia, a series of very severe fits in which there was sub-conjunctival hæmorrhage and numerous bloody puncta under the skin, was followed by steady improvement and ultimate recovery.

In a second, the stupor following an acute attack of insanity seemed likely to pass into permanent dementia, when fits occurred, the patient remaining in a status epilepticus for many hours. From that time she steadily improved, and was discharged well. In the next place, I have met with several cases of general paralysis of the insane in which mental clearing up followed fits. This is the exception; but these cases were clear, and without any other recognisable cause for improvement. I might here say that this point is also made more interesting from the fact that, in some cases of general paralysis of the insane, a bodily illness has once or twice been followed by recession of the acute mental symptoms.

The most striking instance I have to give of mental improvement after fits is the following. A child of a very eccentric father and with neurotic sisters, was so weak in mind, that his education was all but given up. He was never expected to be more than an imbecile, and about fourteen was utterly uneducated. Then for the first time he had fits of an epileptic nature; and from that time he developed, first slowly, and then more rapidly in mental and bodily power, till at eighteen he had attained a good education and lost his fits. He gained an open scholarship, and is now a promising student at one of the Universities.

It is well to remember when speaking of fits, that though there is a common occurrence of fits in general paralysis, yet the two—epilepsy and general paralysis—are very widely separated; and it is strange that, among the many epileptics in a county asylum, very few indeed, if any, die of general paralysis of the insane. I feel I have taken too much space, as it is, with my rather wide wanderings, but I cannot conclude without putting down a few jottings by the way. I think if it be a fact that hallucinations of smell are more common than other hallucinations in epilepsy, some explanation is needed. Hallucinations of smell occur in the insane frequently with sexual disorder, but I see no such relation between epilepsy and sexual perversion.

I think we should study the rate and direction in which destruction of mind due to epilepsy takes. I believe memory is very early and very gravely affected, but that loss of control is the chief effect in the furious cases; so that here we have two groups, one of those tending to dementia, the other those to mania as results of mental losses in different directions. Though impulsive violence is frequently a characteristic of epilepsy, it is not safe to say of every impulsive act done by a lunatic or by an unstable person, that it is epileptic, unless there be a history of fits or of a strongly neurotic family history.

Again; though one chief characteristic of epilepsy is the exact repetition of the symptoms, it does not follow that exact repetition of symptoms connotes epilepsy; as most of us in asylums have met with cases in which the same ideas, following in the same order, give rise to the same delusions and the same insane acts in attack after attack of insanity.

Before winding up, I have a few words to say on general relationships of epilepsy and insanity. Writers have long spoken of an insane aspect, and others of an epileptic facies. There is some truth in both, but not very much, as epilepsy may last for years without leaving any special impress on the features. It has been said that the chronic epileptics assume peculiar mental aspects, and that there are two well-marked groups of these; the first containing the more dangerous class—those who are suspicious and irascible—the other, those who are mild, sheepish. These are, to my thinking, the results of the partial weakness of mind; the one being a state of nervous irritability, easily roused, while the other is weakness and unimpressionability. Epilepsy has for ages been credited with alliance with greatness, and we all know of the great men who have been reputed as epileptics. I would first of all say, that there is little evidence that any of these were really chronic epileptics, the truth being that fits of some sort were present at some period of their lives, and these were all called by one name. It is interesting to find that here, again, we find the alliance between epilepsy and insanity; just as some genius is near insanity, so some fits are met with also in some intellectually great men.

I fear my task now done is but feebly performed; but if it lead to the study and development of these relations and others, I feel sure light will arise, the brightness of which we cannot guess.

ON PSEUDO-TABES FROM ARSENICAL POISONING, WITH A CONSIDERATION OF THE PATHOLOGY OF ARSENICAL PARALYSIS.[1]

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THE two cases which I here present, illustrate the fact that arsenic may cause very typical "pseudo-tabes."[2] The cases also, when studied in connection with recent observations regarding alcoholic, diphtheritic, and plumbic ataxia, make it almost certain that arsenical paralyses are mostly of peripheral origin.

CASE I.

A. C., age 48; single, American.

His mother had a chronic gastralgia of many years' duration. When 20 years old he began to suffer from the same complaint, and as a result became a morphine-taker.

Having reached the point where the amount of morphine necessary to control his pain made him utterly unfit for work, he came to my ward in Bellevue Hospital. At this time, Dec. 21st, 1885, a physical examination showed no organic disease.

Knee-jerk slightly exaggerated, other reflexes normal; urine normal.

On Jan. 1st he was placed on small doses of Fowler's Solution for the relief of his gastralgia, which returned whenever the morphine was withdrawn. The dose was gradually increased until during the whole of April he took ozss. t. i. d. In the latter part of this month the patient developed symptoms of motor and sensory irritation and paralysis involving all the extremities.

There was first a burning feeling in the fingers, toes, and soles of the feet and an itching of the eyes; then a gradual weakness in the limbs developed, with uncertainty in gait and clumsiness and inability to use the hands. He had to give up writing. The burning feelings were followed and supplemented by sensations of numbness and prickling, and occasional sharp pains in the legs. No vesical or rectal trouble was observed. Examination at this time showed the patient to have an uncertain irregular gait. The ordinary tests revealed both static and locomotor ataxia with diminution of muscular sense. He could not stand or walk with his eyes closed, or touch the tip of the nose with the fore-finger, &c. &c. The patellar tendon-reflex was abolished in the right leg; almost so in the left.

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[1] Read at the Annual Meeting of The American Neurological Association, July 21st, 1886.

[2] The term "pseudo-tabes," has been criticised, and with some justice; but it has acquired a wide use and a pretty definite significance in neurology, and so I have employed it.

A patch of anæsthesia existed on the dorsum of the right foot between the first and second toes in the distribution of the *peroneus profundus.* The patient felt as though walking on wool. There was muscular tenderness in legs, especially over motor points. Optic neuritis was observed on ophthalmoscopic examination, kindly made by my friend Dr. Leszynsky. The pupillary reflexes were normal. There were some tremors and muscular twitchings. There was great motor weakness in all four extremities. Dynamometer: right hand 22; left hand 30. The electrical reactions could not be taken until the patient was very much improved. They were then found to be, for the arm muscles, about normal.

For the lower limbs:—

Faradic current: Electrical sensibility greatly diminished.

Electrical contractility, very much diminished.

Galvanic current: Electrical sensibility greatly diminished.

Electrical contractility normal.

Urine normal.

After discontinuing the arsenic the patient slowly improved, but it was five months before all parasthesiæ were gone.

Case II.

The next case was a more striking one. The patient, Jos. B., 29, single, German, varnisher, was kindly sent to my clinic at the N.Y. Post Graduate School by Dr. Wm. M. Leszynsky, whence he was transferred to the hospital.

The patient's parents were both phthisical. Patient himself had had erysipelas nine years ago, tertian ague four years ago, gonorrhœa two years ago. Never had syphilis, was a moderate drinker; not a smoker. Had been in vigorous health up to the evening of March 1st, 1886, when he got drunk and took a handful of Paris green. He vomited all night, and early in the morning he was taken to a hospital, where his stomach was washed out.

For five days he was prostrated and too weak to sit up, but he was not paralysed. About the sixth day he began to feel the sensory troubles, viz. frightful burning pains in his extremities, formication, and occasionally cramps in the legs. Despite this he got up on the sixth day, being then weak, but having no paralysis. About this time he began to develop an ataxia, which he noticed first when he was washing himself. He even found that he tottered and was uncertain of himself when his eyes were closed. The epidermis at this time began to peel from his feet and hands. He had no bladder trouble.

On the 14th day he left the hospital. His strength was increased, but the parasthesiæ and ataxia were unchanged or worse.

He was first seen by me on April 1st, a month after his poisoning.

Examination then revealed the following: General physiognomy: a strongly-built muscular man. His gait is slow and careful. He has to watch the floor, and he brings his feet down hard, the heels striking first. His gait is characteristic of locomotor ataxia.

Mental sphere: his mind is clear, though he is nervous and worried about himself, and he sleeps poorly.

Motor sphere: no absolute paralysis, but much muscular weakness, and he cannot walk up or downstairs, or raise himself on a chair. The paresis is most marked in the extensor cruris of both extremities.

There are no tremors or spasmodic movements.

Dynamometer: right hand 22; left hand 22.

There is great tenderness over the course of the sciatic nerves and over the branches of the brachial plexus in the arm and fore-arm.

Sensory sphere: there is cutaneous hyperalgesia of all four extremities.

Tactile sense, normal; temperature sense, normal. Muscular sense: the patient shows an almost complete loss of muscular sense with consequent ataxia in arms and legs. He cannot tell the position of his limbs, or distinguish between weights differing 1 to 40.

Reflexes: the patellar tendon-reactions are abolished. There is no ankle clonus. Plantar and cremasteric and other skin reflexes are present and normal.

Eye: pupillary reflexes all are normal. Ophthalmoscopic examination by Dr. Leszynsky showed the fundus to be normal; but patient complained of impaired vision.

Mechanical irritations (blows) over motor-points produce no contractions, but striking the body of muscles (t. anticus) produces "muscle waves." On the chest myoid tumors can be raised.

Trophic:—The epidermis has peeled from the feet and hands, once, completely, and a second time partially.

There is some atrophy of the arms and legs.

There are no secretory disturbances. The bowels are regular.

Urine:—At first on admission, sp. g. 1010 acid; clear; no albumen.

One month later (two months after poisoning), sp. gr. 1012 acid; straw colour; trace of albumin (from a gleet?).

Electrical reactions:—

MUSCLES OF LEGS.

Far. current.—No contraction.
E. sensibility lessened.
Galv. current.—ACC almost = KCC.
AOC $<$ KOC.
Contractions sluggish.
E. sensibility lessened.

Thus there was shown to be a partial RD.

Muscles of upper extremities, normal.

The patient improved, but very slowly, the burning sensations giving him most trouble.

He became slightly hysterical in his anxiety over himself.

Cases of the character that I have described are not unknown in literature. Possibly they are not so very rare. The brief descriptions of older writers often indicate that the paralysis approached this pseudo-tabetic form.

Yeesche (*loc. cit.*) reports a case of acute arsenical poisoning, followed a week later by cramps and neuralgic pains in the extremities, paralysis of muscular sense, tactile and pathic hyperæsthesia.

Scolusoboff (*loc. cit.*) notes a case in which there was general atrophy, some paresis, muscular pains and tenderness, loss of muscular sense and hyperalgesia.

Dupuy (*loc. cit.*) reports briefly the case of a patient who, after taking arsenical baths, suffered from parasthesia and diminished sensation in the extremities, "with uncertain gait."

Levin (*loc. cit.*) reports the case of a man, aged 17, who was using an arsenical salve. He suffered from a condition of nervous prostration, followed in 4 or 5 weeks by complete ataxia of all extremities, and a gait which resembled a high degree of chorea.

Seeligmüller (*loc. cit.*) reports the case of a young man who was poisoned by arsenical wall-paper. His symptoms resembled so closely those of locomotor ataxia, that he was declared at first to have that disease. Later the true nature of the trouble was made out. It was distinguished from true posterior spinal sclerosis, by the presence of tendon-reflexes, burning pains in extremities, weakness of extremities, absence of girdle symptoms or vesical trouble.

As to the pathology of arsenical poisoning, authorities differ. In the papers of Da Costa, Mills, and Seguin, published four and five years ago, the view was held that the anatomical change was a diffuse myelitis.

Da Costa thought it more of an anterior poliomyelitis. Others have seen in it a resemblance to the subacute spinal paralysis of Duchenne. German authorities (Strümpell, Leyden) have rather inclined to the view that the disease is a neuritis.

No autopsies have yet been recorded. The only reference to such is that of Wibmer (*loc. cit.*), who says that the lower part of the cord is always affected, as is shown by the frequent congestion of the cauda equina.

Apart from the clinical history, the most conclusive evidence of a myelitic origin was thought by Seguin to be the experiments of Popow, published in 1881, upon rabbits.

Kreyssig, a year ago, however, working under Schultze of Heidelberg, found in the spinal cords of healthy rabbits all the changes which Popow described as being present after arsenical poisoning. On the other hand, in six rabbits poisoned with arsenic, he found no pathological changes in the cord.

Pistorius states that the nervous system of rabbits and guinea-pigs is very sensitive to arsenic, while that of cats and dogs is less so. It is very evident, therefore, that no pertinent conclusions could be drawn from experiments with rabbits, even if there were any such data to start with, as Popow believed that he had. Jaeschke gave a dog arsenic in large doses for ten days, then gave a fatal dose. Paralytic symptoms, though not well marked, developed. On post-mortem he found a few small apoplexies in the spinal meninges, but nothing to indicate a beginning myelitis.

At present, therefore, we must rely upon the clinical history of the cases to determine the pathology. But the evidence, it seems to me, here is so strong as to amount almost to a demonstration. One cannot read over the histories of cases of arsenical paralysis without observing how closely they tally with those of multiple neuritis, as described in systematic textbooks, like that of Strümpell.

Jaeschke (*loc. cit.*) who, in 1882, reported two cases of arsenical paralysis, argued that the symptoms were due to neuritis, because—

1st. Of the localization of the sensory or motor disturbance in the limits of certain nerves. In one of his cases there was a well-marked anæsthesia, limited to the regions of the median nerve.

2nd. The marked sensory disturbances, especially the peculiar burning pains, ataxia, and tenderness along the nerves.

3rd. Absence of the characteristic symptoms in profound spinal paraplegia, viz. of vesical weakness.

4th. Absence of the rapid and high grade atrophy which follows anterior poliomyelitis.

5th. The comparatively rapid and complete cure, despite a high grade of paralysis.

6th. The presence of herpes zoster.

7th. The electrical reactions, which are those of a mild form of peripheral paralysis.

8th. The marked tenderness of the paralysed muscles.

A. To these arguments I would add that of the presence of optic neuritis, as in Da Costa's case and one of my own.

B. The occasional arsenical monoplegias, much as that described by MacKenzie of one of the vocal cords.

C. The fact that arsenic may produce a perfect type of pseudo-tabes, like that from alcohol or diphtheria, and the fact that in these latter cases the trouble has been shown to be a neuritis.

I beg to enlarge upon this latter point.

It is very well known that there are post-diphtheritic paralyses of various types, and that among these is a form of ataxia or pseudo-tabes due to the diphtheritic infection. It was noticed first by Orillard, in 1836, and differentiated by Eisemann, and Jaccoud later. Many illustrations of it have been reported, and Buzzard, in his work, acknowledges that it may even be mistaken for true tabes. The only post-mortem made in a case of this kind was that of E. Mendel.[1] He found a well-marked neuritis, with slight central changes, indicating, perhaps, a beginning myelitis. The post-mortems made in other and ordinary forms of diphtheritic paralysis have shown, with very few exceptions, well-marked neuritis, with slighter central changes. Déjérine's cases are the most important in contradiction, and he found degenerative neuritis as well as myelitis.[2] On the other hand, Charcot, Vulpian, Lorain, and Lépine, Liouville, Leyden, P. Marie, Buhl, P. Meyer, and E. Gaucher, have all found decided evidence of neuritis.[3] So that it may be said with much confidence that the diphtheritic pseudo-ataxia is of peripheral origin.

Again, there is known to be an alcoholic paralysis proper and an alcoholic pseudo-tabes. The latter disorder closely resembles the pseudo-tabes I have described. It is very well settled that both these forms of alcoholic paralysis are due to a neuritis. The evidence is fully gone over in the articles by Dreschfeld[4] and by Schultz.[5] It is very probable, also, that in the rare cases in which lead causes pseudo-tabes the disturbance is a peripheral one.

That a pseudo-tabes may be caused by neuritis is shown again in a case reported by Déjérine.[6]

Several have described arsenical paralysis as resembling the subacute spinal paralysis of Duchenne. But Leyden cites two cases with post-mortem, one of Eisenlohr, and one of his own, to show that this is a multiple neuritis.

There are, then, two forms—often not very sharply defined—of arsenical paralysis, just as there are of the post-diphtheritic and alcoholic paralyses, viz. (1) arsenical paralysis proper, (2) arsenical pseudo-tabes. Clinically these various toxic paralyses have a close resemblance; indeed, arsenical pseudo-tabes is as nearly identical with alcoholic pseudo-tabes as well can be. Now since the latter is known to be due to a peripheral lesion, it is a legitimate inference that the former is so also. Until post-mortems are obtained we cannot get any nearer certainty.

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[1] 'Centralbl. f. Neurol.,' 1885, No. 6.

[2] 'Arch. de Phys.,' p. 107.

[3] *Vide* E. Mendel's article, *loc. cit.*

[4] 'BRAIN,' Jan. 1886.

[5] 'Neurol. Centralbl.,' No. 21, 1885.

[6] 'BRAIN,' 1884, p. 558.

We have at least shown, however, how entirely improbable is the dictum that arsenical paralyses are always due to a diffuse myelitis, although in some cases such lesion may exist. And we have shown another cause for peripheral tabes. The establishment of the fact, that arsenic as well as alcohol, diphtheria, lead, and probably other poisons and infections, may cause a disease closely resembling posterior spinal sclerosis, will do much to clear up the doubts that have of late attached to the unity and autonomy of that disease. Cases of locomotor ataxia reported cured, and cases of supposed posterior spinal congestion will have to be reviewed in the light of these facts. When Déjérine described as locomotor ataxia a case which now appears to have been one of alcoholic peripheral neuro-tabes; when Buzzard has diagnosticated as true spinal tabes a case of post-diphtheritic ataxia; and when Seeligmüller mistakes for a time a case of wall-paper poisoning for one of true spinal tabes, we may easily suppose that errors have been made by many others, especially those who have been so fortunate as to see patients with tabes cured.

CONCLUSIONS.

I. That a disease resembling locomotor ataxia may be caused by arsenic, given medicinally, absorbed from wall-paper or taken in a single dose.

II. That arsenical paralyses of this type, and arsenical paralyses of other types, are not due, as a rule, at least, to a diffuse myelitis, as has been taught, but to a multiple neuritis.

III. That arsenical paralyses, like those from diphtheria, alcohol, lead, and probably other infections and poisons, are of two types.

(*a.*) The ordinary mixed motor and sensory paralysis; the motor troubles and atrophy being more marked.

(*b.*) The pseudo-tabetic form in which there is no pronounced motor paralysis, but marked sensory troubles, and especially ataxia.

BIBLIOGRAPHY.

As arsenical paralysis is not yet systematically treated of in text-books, and as no complete bibliography has been compiled, I have undertaken to collect the references. The task is made comparatively easy through the learned contributions of Imbert Gourbeyre, who, in a series of articles published in the 'Gazette Médicale de Paris,' 1858, and in an elaborate monograph, entitled "Des Suites de l'empoisonnement par arsenic," Paris, 1881, covered the ground quite thoroughly up to five years ago.

Arsenical paralysis was first referred to in the 14th century by P. Albano: "De Venenis eorumque Remediis." The fact that arsenic in toxic doses may cause paralysis was evidently known to most medical and toxicological writers who succeeded him, and was referred to by Forestus, 1550; Kircher, 1578; Weikard, 1626; Schroeder, 1641; Ramlovius, 1650; Tirnocus, 1662; Wessfer, 1629.

In the 18th century we have the following references:

Ramazzini: 'De mortis opificum,' 1703.

Blancard: "Deux Cas d'emp. par ars."—'Opera med. chir.' 1701.

Dehaën: 'Ratio medendi,' pars ix., c. vi., 1763—case with total desquamation of epidermis of feet.

De Boissieu: 'Mémoire sur les méthodes rafraîchies,' Dijon, 1772.

Barrier: 'Journ. de Méd.,' 1783.

Pyl: 'Aufsätze u. Beob.,' viii., Berlin, 1784.

Hahnemann: 'Ueber Arsenik-Vergift.,' Leipzig, 1786.

Geoffroi, in 1741, first wrote systematically of the affection.

Cranz, in 1755, and Hahnemann also described the symptoms quite thoroughly.

In the 19th century the same class of reports was made, there being no careful attempts at analysing the symptoms, or searching out the true pathology, until within the past twenty years. The chemical and forensic sides of the subject have almost always been in the foreground, and no post-mortems are reported. I have used the abbreviations Chr. p. q. o. and l. o. for chronic paralysis, quick or late onset.

Metzger: 'Med. gericht. Abhandl.,' Königsberg, 1804. Chronic paralysis coming on late, i.e., four to fourteen days after poisoning.

YELLOLY: 'Edinb. Med. Journ.,' 1809. Acute, rapidly coming on, death in 33 hours.

PHILENIUS: 'Medic. Chirurg. Bemerk.,' Frankfort, 1809. Chronic paralysis, rapid onset.

BERNHARDI: 'Annalen der Heilkunst,' 1811. Chr. par. q. o.

BACHMAN: 'Abhandl. der Med. Phys. Gesellsch. zu Erlangen,' 1812, and 'Magazin f. Med. Chirurg. und Pharm.,' Aarau, 1816. Chr. par. l. o.

REMUR: 'Horn's Archivs,' 1813. Caused by medicine.

EBERS: 'Journal de Hufeland,' 1813.

SCHÆFFER: Ibid., 1816.

HARDEGG: 'De vario Arsenici in animalia effectu,' Tübingen, 1817. Ch. p., late onset.

BERNT: 'Beiträge zu Gericht. Arz.,' 1818.

BORGES (C. F.): 'Rust's Magazin,' 1819.

BEYER: 'Horn's Archivs,' 1820. Chr. p. q. o.

MURRAY: 'Edin. Med. and Surg. Journ.,' 1822. Ch. par. q. o.

COOPER (Sir A.): 'Lancet,' 1823. Chr. p. l. o.

LEURET: 'Recueil périodique,' 1826. Chr. p. q. o.

SCHEGEL: 'Journ. de Hufeland,' 1827.

WIBMER: 'Die Wirkung. der Arz. im Gift,' München, 1831–40.

STACHOW: 'Henke's Zeitschr.,' 1833. Chr. p. q. onset.

BUZORINI: 'Würt. Med. Cor. blatt,' 1835. Rapid onset, transitory.

LACHÈSE: 'Annal. d'hygiène,' 1837. Chr. p. q. o.

HAFFTER: 'Schweiz. Zeitschr.,' 1839. Sudden; fatal in twenty-seven hours.

ORFILA: 'Archives Générales,' 1839; also 'Traité de Toxicologie,' ch. p. l. o.

KELLERMANN: 'Oester. Jahrbuch,' 1840. Ch. p. l. onset.

SERPH: 1841, 'Med. Chir. Rev.' Transitory, acute, paraplegia.

ZOLLNER: 'Medic. correspond. Baier. Aerzt.,' 1841.

OPPLER: 'Med. Zeit. in Preussen,' 1841.

FLECHER: 'Verhandl. der k. k. Gesellsch. der Aerzt. zu Wien,' 1843.

SHIPMAN: 'American Journ. Med. Sciences,' 1843. Ch. p. l. o.

JONES (R.): 'Provincial Med. and Surg. Journal,' 1844. Caused by medicine.

CHRISTISON: 'Treatise on Poisons,' 1845, p. 315.

WOODCOCK: 'Lancet,' 1845. Chr. p. q. onset.

RUMPELT: 'Henke's Zeitschr.,' 1846.

SCHAPER: 'Beiträge zu der Lehre von der Arsenikverg.,' Berlin, 1856. Ch. p. l. o.

BASEDOW: 1846, Paralysis from wall-paper.

BISSELL (E.): 'Amer. Journ. Med. Sciences,' 1848. Sudden onset, acute course.

CLARK: 'Bost. Med. and Surg. Journal,' 1848. Ch. p. l. o.—Same? 'N. Am. Hom. Journ.,' 1851.

SPENGLER: 'Henke's Zeitschr.,' 1848. Acute, sudden onset.

GEOGHEGAN: 'Dubl. Med. Press,' 1850. Remarkable desquamation.

COTTON: "Arsenical paralysis."—'New York Journ. of Medicine,' 1850.

MACCREADY: 'Amer. Journ. Med. Sciences,' July 1851. Acute fatal.

SKILLTON: 'Bost. Med. and Surg. Journal,' 1851.

DEVERGU: Paris, 1852. Right hemianæsthesia, transitory.

ARAN: 'Union Médicale,' 1852. Ch. p. l. o.

HUSS, M.: 'Chronisch. Alcoholisch. Krankh.,' Stralsholm, 1852.

GOEPPERT: 'Henke's Zeitschr.,' 1852.

GOULLON: 'Allgemein. Nom. Zeit.,' 1853.

CHRISTISON: 'Edinburgh Med. Journ.,' 1856.

YEESCHE: 'Hom. Viertel von Cl. Müller,' 1857. Pseudo-tabes, chr. p. l. on.

L. D'ETIOLLE: 'Gaz. Hebdom.,' 1857.

D. GOURBEYN: 'Gaz. Méd. de Paris,' 1858, and Paris, 1881.

TREUD: 'Brit. Med. Journ.,' 1858. Arsenic taken to cause abortion.

TAYLOR: 'On Poisons,' 1859.

GIBB: 'Trans. Path. Soc. of London,' 1860. Caused by medicine.

KRAUS: Liège, 1862. Chr. p. q. o.

MACKENZIE, M.: 'Annuaire Cavasse,' 1862. Laryngeal paralysis (left vocal cord).

KEBER: 'Vierteljahrb. f. Gericht. Med.,' 1863. Ch. p. l. o.

SMOLER: 1863, 'Oester. Zeitsch. f. pract. Heilk.,' ix. Nos. 19, 20.

JACCOUD: 'Les Paraplégies et l'Ataxie du mouvem.' Paris, 1884, p. 323.

BELL: 'Lancet,' 1866. Ch. p. q. onset.

DODD: 'Lancet,' 1866. Ch. p. q. onset.

ZELLER: 'Würtzb. Corresp. bl.,' 1866. Ch. p. l. o.

BOLANDER: 'Schmidt's Jahrb.,' Bd. 145, 1867.

LOLLIOT: 'Thèse de Paris,' 1868. Chr. p. q. o.

GRAHAM: 'Glasgow Med. Journal,' Nov. 1868. Schmidt's Jahrb. Bd. 145.

ISIDORE AND EICHINGER: 'Recueil des Mém. de Médecine milit.,' 1868. Ch. p. l. o.

DUPUY: 'Gaz. des Hôp.,' 1868, p. 145. Virch. 'Hirsch Jahrb.,' 1868.

WORMLEY: 'Medical Times and Gazette,' 1869. Quick onset; fatal 36 hrs.

KIRSCHGASSER: 'Brit. Med. Journal,' 1869. Poisoning from wall-papers.

Seisser: 'Bayr. Intellig. bl.,' 1869. Chr. par. l. o.

Ferrand: 'Union Méd.,' 1872. Chr. p. l. on.

Malmsten: 'Hygeia,' 1873. From wall-paper. Also 'Schm. Jahrb.,' Bd. 165.

Levin: 'Hygeia,' Bd. xxxv., 1873, and 'Schmidt's Jahrb.,' 165.

Taylor: 'Manual Med. Jurispr.,' New Ed., 1873.

Gaillard: 'Société de Méd. Légale de Fr.,' t. 3, 1874. Caused by medicine.

Scolusoboff: 'Gaz. Méd.,' 1875. Chr. p. l. on. pseudo-tabes?

Nicolaysen; 'Schmidt's Jahrb.,' 1875. From wall-paper.

Hohn: 'Schmidt's Jahrb.,' 1875. From wall-paper.

Brown: 'The Sanitary Record,' Boston, 1876. From wall-paper.

Eckstein, S.: 'Ueber Atrophie u. Paralys. nach Acut. Arsenik Intox.' Thesis, Breslau, 1876.

Popow: "Ueber die Verander. im Rückenmarke," &c.—'St. Petersburg. Med. Woch.,' 1881, No. 36.

Da Costa: 'Phil. Med. Times,' March 1881.

Seeligmüller: "Ueber Arseniklähm."—'Deut. Med. Woch.,' 1881, No. 14. 1 case pseudo-tabes.

Seguin: 'Journ. Nerv. and Mental Dis.,' Oct. 1882.

Gerhardt: "Ueber Arsenik muskul. Atrophie," &c.—'Sitzung. der Phys. Med. Gesellsch. zu Würzb.,' 1882, p. 28.

Jaeschke: 'Ueber Lähmung nach Arsenikverfg.'—Thes. Breslau, 1882.

Mills (C. K.): 'Journ. Nerv. and Ment. Dis.,' Jan. 1883.

Eiselt (T.): 'Medico-chirurg. Centralb.,' Wien, 1883, xviii. 324. Ein Fall von Arsenik paral.

Burton: 'Lancet,' June 28, 1884.

Freyssig: 'Virchow's Arch.,' Bd. 102, 1885. "Ueber der Beschaffenh. des Rückenm. bei Kaninch. u. Mund. nach Phosphor u. Arsenikvergift."

The above bibliography includes over 100 references.

Among 120 cases collected by Gourbeyre, the paralysis in over one-half was cheiro-podal, stopping at elbow and knee. In $\frac{1}{4}$ there was paraplegia. In 4 cases hemiplegia. In 2 cases hemianæsthesia. In 3 cases of one limb. In one case of a vocal cord. Sensory troubles are always present, and precede the motor. Anæsthesia may follow in distribution of a nerve. Burning, and lancinating pains, and great muscular tenderness are characteristic. Desquamation of the epidermis often occurs. Herpes zoster has been noticed. Atrophy is often general, but in chronic cases may affect special groups of muscles. Almost all cases recover.

BIBLIOGRAPHY OF CHEMICAL INVESTIGATIONS.

Chemical studies, in order to discover the distribution of arsenic in the body of persons or animals dying from arsenical poisoning, have been made by—

Scolosuboff: 'Bulletin de la Société Chim. de Paris,' 1875, p. 24.
Ludwig: 'Wien Acad. Anzeiger,' 1879, p. 181.
Johnson and Chillinden: 'Am. Chem. Journal,' vol. ii. p. 332.
Vaughan and Dawson: 'Journal of American Medical Association,' 1884, p. 152.
Enareschi (J.): 'Rivista di Chem. Med. e Farm.,' 1883, p. 17.

And several others quoted in Jaeschke's 'Thesis.'

The original view put forth by Scolosuboff, that there is a great proportional excess of arsenic in the brain and cord, has been abundantly refuted by many other observers (see above). It is now well determined that, in almost all cases, arsenic is absorbed by the different organs in about the order named: the liver, spleen, intestines and kidneys take up the most; the brain generally has only a trace, sometimes none at all, while the muscles take up a considerable amount.

COMA.[1]

BY CHARLES MERCIER, M.B.

In one of the latter numbers of the 'Medical Times,' Mr. Savory had an important and suggestive note calling attention to the vagueness of meaning which attaches to the term *coma*, and to the pathological and clinical diversity of the conditions to which the term is applied; and he sought to restrict its application to a particular class of cases. He would have the term *coma* associated with those cases only in which there is a state of insensibility from which the patient cannot be completely aroused, together with a tendency to death chiefly by asphyxia.

To me, this limitation proposed by Mr. Savory appears strictly scientific and thoroughly practical: scientific because these two conditions—insensibility and tendency to asphyxia—are invariably associated with other morbid conditions in a well-defined group, so marking a state as definite pathologically as it is clinically; and practical because the state thus characterized may invariably be recognised by a test that it is extremely simple and absolutely pathognomonic, being never absent in coma, and never present except in coma as thus defined.

Looking at the matter from a point of view different from that of the surgeon, I would substitute for Mr. Savory's expression, "insensibility from which the patient cannot be completely aroused," the expression "evidence of defect of consciousness." The meaning of the latter expression will doubtless be much less obvious than that of the former, but it is for the present purpose more convenient. When we say that a person is insensible, we make a statement which is scientifically unjustifiable. All that we are warranted in stating is that he gives no evidence of consciousness. To insist on accuracy in this matter is not mere pedantry, for those who are familiar with cases of insanity know that many people have appeared to be, and have been recorded as, "insensible," "unconscious," "not knowing what is going on around them," and so forth, when, as a matter of fact, they are fully alive to passing events, and were able subsequently to describe them.

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[1] The descriptive portion of this article appeared in substantially the same form in the 'Medical Times' for March 28th, 1885. The pathological discussion is new. Both portions are inspired by the teachings of Dr. Hughlings-Jackson, who must be credited with whatever of value the article contains.

The reason that the phrase "defect of consciousness" is substituted for "insensibility," is that the latter implies absolute unconsciousness; it is used convertibly with "loss of consciousness;" it implies temporary mental annihilation. But in very many cases of coma, while there is evidence that much consciousness is lost, there is also evidence that some consciousness remains. Such terms as "insensibility" and "loss of consciousness" imply and suggest that there is an abrupt separation between full mental efficiency and total mental nothingness, and that a person must be in either the one state or the other. But this is not the fact, and the phrase "defect of consciousness" seems better adapted to convey the notion, that between the full daylight of vivid mental activity and the utter darkness of unconsciousness there is a long twilight, in which consciousness is not wholly lost, but is defective in various degrees. Just as on the physiological side there is every gradation from full mental alertness through fatigue, lassitude, drowsiness, semi-somnolence, sleep with vivid dreams, sleep with faint dreams, to profound slumber; so on the pathological side there is every gradation from slight confusion, through dulness and many grades of coma, to deep "insensibility."

To Mr. Savory's doctrine that, in that state to which the term coma should be limited, this evidence of defect of consciousness is always associated with a tendency to death by asphyxia, I entirely agree; but again, looking at the matter from another point of view, this tendency to death by asphyxia —this failure of the respiratory process—appears but a part of the bodily condition with which the defect in consciousness is associated. In deepest coma, when there is "insensibility" so profound that no amount of stimulation evokes any answering reaction, there is laboured respiration, and there is much besides. The respiratory defect is peculiar. There is a deep inspiration, followed by a long noisy expiration, and then a long pause. In other words, the respiratory centre does not act with its normal freedom. The small accumulation of carbonic acid, which is normally enough to excite its action, is no longer enough. A stronger stimulus is required. The effete ingredients must accumulate for four or five times their usual period before they reach the degree of concentration which can act as a stimulus to the sluggish centre. When the respiratory act does occur it is imperfect. Only the fundamentally necessary parts of it, the raising of the ribs, is well performed. All the accessory portions are imperfect, and the less essential they are, the more imperfectly are they performed. The diaphragm does not descend to an extent at all proportionate to the elevation of the ribs. The loud gasp that occurs even when the mouth is open and the tongue pulled forward, shows that the glottis does not dilate properly. Flaccidity of the soft palate allows the loud snore; imperfect action of the lips permits the purring sound. It has been said that the respiratory defect is but a part of the bodily condition. The skin is relaxed; it is flaccid and covered with moisture. If we pass a catheter the urine flows sluggishly; the bladder does not contract vigorously. Throw a bright light upon the eyes: the pupils will contract little or not at all. The pulse is very full; in other words, the arteries are flaccid—their muscular coat is relaxed. But there is much more than this. *The limbs are all paralysed.* It is generally supposed that the patient lies still because he is unconscious; but, as Dr. Hughlings-Jackson, in his Croonian lectures, has shown, this is nonsense. It is the commonest thing in the world for an unconscious person to move his limbs. The fœtus in utero moves its limbs before consciousness has come into existence. But no one, conscious or unconscious, can move a paralysed limb. The comatose patient lies still, it is true; but examination shows that the limbs are not the limbs of a person who lies still because he does not want to move. The muscles are flaccid. If we raise a limb and drop it, it falls like the limb of a corpse. It flops down in a flail-like manner that does not occur in, and cannot be imitated by, a limb that is not paralysed.

Similarly, the head rolls from side to side under the influence of gravity, without check from any tone in the muscles of the neck. To sum up the bodily condition of the comatose patient: there is paralysis, more or less complete, of all the voluntary muscular system; there is incomplete paralysis of the muscular system of the skin, of the arteries, of the bladder, of the pupil; there is incomplete paralysis of the respiratory system; and the muscles of the heart appear to be the only ones that are not weakened in some degree.

If we examine patients whose coma is less profound, we obtain evidence of some remnant of consciousness. On violent and repeated irritation, the patient feebly moves a limb; he groans; under a strong light his pupils sluggishly contract; his face is less dusky; his pulse is less full; his diaphragm acts better. But he still snores; he is still incapable of strong movement; he cannot hold up his head. In this case there is some evidence of dim consciousness, and there is paralysis of less degree than in the previous case.

Patients who exhibit a less degree of coma are more easily aroused. Manifestations of consciousness occur under smaller stimuli, and are of more vigorous character. Pinch the skin and they swear; repeat the pinch and they strike out; throw a light into the eyes and the lids close. But their movements are feeble and are not sustained; they cannot sit up; they do not answer the simplest question; they cannot lift hand or knee against a feeble pressure. There is still evidence of defect of consciousness, and there is still paralysis.

There is a stage of mental confusion and of bodily weakness which is not coma—is not severe enough to be called coma—but is a still slighter degree of the same malady. In such cases the patient can talk, but he talks stupidly. He can answer simple questions, but is easily confused. He can stand, but he sways about. He can walk, but he reels. Here, again, there is evidence of defect of consciousness, and there is muscular weakness—there is some degree of paralysis.

The four stages described are connected by innumerable intermediate gradations, rendering the whole series continuous; but to whatever degree the coma has extended, the amount of paralysis present is always proportionate to the degree to which consciousness appears defective. The bodily defect and the mental defect proceed *pari passu,* and signify, it cannot be doubted, the same lesion. The various stages of coma may all be necessarily exhibited in the same individual, and are so exhibited to perfection in the coma due to alcohol.

It is to be particularly noted, and this observation is due to Dr. Hughlings-Jackson, that the paralysis is first a loss of *movements,* and only at last a loss of *motion.* That is to say, the finest, most delicate, and most elaborate movements, and those that are most associated with Will, are the first to go; while the simplest, broadest, most general movements, and those least associated with Will, are the last to be retained. It is under the operation of this law that the drunkard first becomes unable to write; then his articulation gets thick; then he spills his liquor; then he is unable to walk, and at length sinks under the table. But this is not all; *it is under the operation of precisely the same law that his breathing begins to fail, while his heart remains unaffected,* and that, as has been pointed out, the accessory parts of the breathing apparatus fail before the fundamental parts. The full pulse and the stertor are all of a piece with the inability to write and the thick articulation. They are due to an extension of the same process in the same direction in the same way; and this is why it is said that those cases of insensibility in which the tendency to death is primarily by asphyxia form a natural pathological group. To say that the tendency to death is chiefly by asphyxia is to say that the breathing is failing before the heart; and this is the natural termination of the long process of degradation which begins with the highest and most elaborate movements and spreads disastrously and continuously down to the lowest, simplest, and most fundamental. Follow the process to the bottom, and you find that the last point of attack must be the heart, and the penultimate point the breathing.

I have said that there is a test for coma which is absolutely pathognomonic. This statement must be understood to exclude the pre-comatose stages of coma, and to apply to those stages only in which there is what is ordinarily termed "insensibility"—in which the patient is incapable of standing, or sitting, or answering questions; although it occurs in very light coma, and sometimes before the conjunctivæ become insensitive. The test to which I refer is one to which attention was drawn—as a symptom—both by Dr. Warner and myself, in the 'British Medical Journal,' about eight years ago. It is the movement of the two eyes independently of one another. About the time that the conjunctiva becomes insensitive, the eyes are noticed to have a peculiar unintelligent "far away" expression. If examined, it is found that this expression is due to the absence of any convergence to their axes. They are quite parallel. The next thing is, that they begin to move independently. Usually one rolls out while the other remains still. Granting the absence of previous strabismus, actual divergence of the eyes is absolutely certain evidence of coma. There are people who can move one eye through a small definite arc while the other remains fixed, but no one can voluntarily cause his eyes to diverge; and in coma the eyes, if watched, will *always* be seen to diverge. They do not always begin by diverging; they may converge; they may both move in the same direction—upwards or downwards, to right or to left—but they will not both move at the same rate and to the same extent. The deeper the coma becomes, the more independent become the movements of the eyes. The difference in their direction is often astonishing. One may be directed out and up, and the other out and down. One may be directed downwards, while the other is hidden beneath the upper lid. If the patient can be momentarily roused by severe stimulation, the eyes momentarily resume their parallelism; as he subsides into coma, so they fall back into independence. It matters not what the cause of the coma may be—whether alcohol, ether, chloroform, uræmia, hæmorrhage, a previous epileptic fit, a blow on the head, meningitis, cerebral tumour, or what not—if there is coma the eyes move independently; if the eyes move independently there is coma. I am unable to say whether the same thing occurs in normal sleep, but it very likely does occur, sleep being physiological coma. From a diagnostic point of view, the importance of this symptom is evident. If only because it instantly negatives the possibility of hysteria, or malingering, it should always be looked for in light coma. So easy is it to recognise, that I have long been in the habit of teaching nurses and attendants to look for it, and to regard it as the first sign of danger.

Such being the clinical phenomena, the question for us is, What is the pathological process that these phenomena indicate? What are the changes inside the body to which these outward and visible signs are due?

Two characteristics of the phenomena are specially to be noted. First, that there are two sets of changes; and second, that these two sets of changes proceed concurrently. There are the bodily changes, and there are the mental changes. Difficult as it usually is to establish a parallelism between bodily and mental phenomena, here the parallelism is obvious. Both sets of changes are in the same direction. Both are in the direction of *enfeeblement*. Consciousness—so far as we can judge of it by its outward manifestations, and by the descriptions of those who have gone through the process—marches steadily from the light, through a penumbra of greater or less breadth, into the darkness of oblivion. As with the mental strength, so with the bodily strength. There is a continuous loss of muscular power, ending in complete powerlessness. The slight unsteadiness becomes a reel, the reel ends in a fall, the struggles subside, and the patient lies like a log, the picture of utter prostration.

There is a still further parallelism, and a very remarkable one. The enfeeblement, both in the one case and in the other, is not a mere enfeeblement. It proceeds not at random, but in an orderly march, obliterating one faculty after another, one function after another, in regular sequence. Ask a man who is just leaving a convivial dinner to settle with you the lease of his house, or a deed of partnership. He will naturally and certainly refuse. If you press him, he will say it is not a proper time; and if pressed further, will explain that to take him now is unfair, for to such an important and delicate matter one must come with a clear head. The admission is, that the mind now is not as vigorous as it will be to-morrow morning. It is slightly enfeebled. In other words, by the fatigue of the day—by taking a heavy dinner, and so drafting off a portion of the blood-supply from his brain to his stomach, by imbibing alcohol, and so benumbing to a certain extent his highest nervous centres—*he has travelled the first stage on the road to coma.* Now note carefully the character of the enfeeblement. Our *convive* is still able to enjoy his chat and his joke, to pay his legal cab fare, and to scold his wife; but he will not trust himself to settle the precise terms of his will. What he is unable to perform are *operations of a high degree of complexity, delicacy, and precision.* Similarly on the bodily side. He can walk perfectly straight, he can light a cigar without bungling, and button his overcoat with ease; but when he tries to play billiards he finds his "hand is out." So that of bodily movements, as well as of mental operations, he has lost the most precise, the most delicate, the most complex.

As the coma deepens, the parallelism is maintained at successively lower stages. When the drunkard has reached that stage at which his articulation is thick and his words run into one another, he has on his mental side reached a stage of confusion of mind such that he no longer appreciates that the stake he is playing for is beyond his means. When the invading feebleness reaches his locomotor movements, and, overcome with languor, he staggers to a couch, his mental insufficiency has so increased that he mistakes the room for his bed-room and drops his boots into a cupboard in the belief that he is putting them outside his door. By the time that his bodily weakness has reached the stage of prostration, so that he is unable to rise, and by his utmost effort can only kick or strike out aimlessly and feebly, his mental capacity has reached a corresponding stage of diminution, he has lost all knowledge of his surroundings, and is merely confusedly conscious of the discomfort of being disturbed. When the prostration is complete, consciousness is lost.

Having regard to the strict and constant parallelism between the two sets of changes, the inference is unavoidable that they are due to the same lesion. It is infinitely improbable that two separate lesions should begin at the same moment, follow precisely the same course, step by step keep time together, and culminate simultaneously.

Whatever the intimate nature of the lesion, the nature of the phenomena—their constant character of enfeeblement—indicates that the lesion must be of an enervating character: it must have on the functions of the nervous centres a weakening, arresting, obliterating, nullifying influence.

Seeing that at the very outset the most elaborate mental processes are affected, the starting-point of the lesion must be in that nervous region whose activity underlies these processes. Seeing that at the outset the most elaborate bodily movements are affected, the starting-point must be in that nervous region whose activity actuates these movements. Hence it appears that the highest of all nervous regions, viz., that which underlies the most elaborate mental processes, represents at the same time the most elaborate bodily movements. So that when the highest of all nervous processes are arrested, by this single pathological event is produced the double set of symptoms—the enfeeblement of consciousness and the enfeeblement of conduct.

At the next stage of the malady, the further enfeeblement of consciousness and the concomitant and parallel enfeeblement of movement indicate an extension of the original lesion. They indicate that, in addition to the loss of the highest nervous processes, there is now a loss of other and subordinate processes of a less elaborate and more fundamental character. As the lesion extends, all those nervous regions are at length put out of action which subserve, however faintly, the manifestations of consciousness; and consciousness is altogether extinguished. By the same lesion all movements classed as intelligent are obliterated. Should the erasing process descend still further in the nervous centres, it lays hold at length upon the last strongholds of vital activity. It stills the breathing; it stops the heart. Thus the whole double train of symptoms displayed in coma are accountable for by the gradual extension of a single lesion.

But the train of symptoms in coma is not merely double.

It is multiple. And each train of symptoms runs a similar parallel course, and exhibits the same series of gradations as those we have already considered. Herein lies an implication of profound significance.

At the very outset of the malady, at the very first step of the long journey which terminates in coma at the door of death, not only is there some enfeeblement, however slight, of mind, not only is there some enfeeblement of body, but there are other phenomena also. The face is flushed, showing diminished vascular tension; the eye is bright, showing greater fulness and increased secretion; the pulse is more rapid, showing altered cardiac innervation; the skin is moister; the renal secretion is augmented; the stroke at billiards, which now fails, requires not only a precise adjustment of ocular muscles and a precise movement of hand and arm, but also a firm attitude of legs, and a steady tension of the muscles of back and belly. At the very outset of the coma change there is an affection not only of the mind, not only of the most elaborate movements, but also *of every part of the body.* The entire organism is affected. And this affection is due to a lesion of the highest of all nervous processes. Thus we arrive at this most important conclusion: that the highest nervous processes, which form the substrata of the most elaborate mental operations, represent at the same time not only the most elaborate forms of conduct and muscular movements, but also *every part of the organism* in some degree.

It may be objected, that in the case supposed—that of incipient coma due to alcohol poisoning—the similar effects on various functions may be produced separately by the direct action of the alcohol upon various separate centres,—the vaso-motor centre, the cardiac-inhibitory centre, the renal secretory centre, the cutaneous secretory centre, and, for aught I know, the oculo-glistening centre and the billiard-playing centre. But this explanation will not hold water, for it will not account for the strict parallelism of the involvement of so many separate functions, nor will it account for the progressive and parallel involvement in coma from such a cause as cerebral tumour.

Moreover, we can reach the same conclusion by two other very different routes; and the fact that we can do so, goes far to place the conclusion beyond question.

The first route is this. The highest nervous centres are the anatomical substrata of consciousness; and consciousness *in toto* is made up of object-consciousness and subject-consciousness—of that which is thought and that which thinks. Of object-consciousness, the physiological substratum or condition is the reverberation of the nervous currents debouching into the highest centres from the organs of sense, and so from the external world. Of subject-consciousness, on the other hand, the physiological substratum is, as Ribot has pointed out, the wash on the shore of the highest nervous centres of nerve-waves arising from the interior of the body. And if from the body, then necessarily from the whole body, or the subject-consciousness would be partial—erroneous, as indeed it becomes when, by the severance of a nerve-trunk, the currents flowing from any part of the body are arrested.

The second route is more direct. Let it be granted, that the function of the highest nervous centres is to adjust the organism as a whole to its environment; then must the whole organism be represented in these centres, or the adjustment will be erroneous—will be not that of the whole organism, but of a part only.

The establishment of this principle—that the highest nervous centres represent in more or less degree every part of the organism—is momentous, and leads to results of the very highest importance.

To the physiologist it is of value, since it demonstrates that the various functions of the encephalon are not segregated in separate encapsuled portions of the grey matter, and shows the inadequacy of that view which regards one localised portion of the brain as representing movements of the arm only, another of the leg only, and so forth. The doctrine of nerve-centres is rapidly becoming, like so many doctrines before it, a fetishism. Some years ago every obscure symptom was accused of being "reflex," and reflex action was regarded as the universal solution of every clinical problem. Reflex action was displaced by incoördination—a term which by its superior vagueness and sonorousness was still better qualified for the post of Universal Cause. But incoördination has been deposed in its turn and beaten clean out of the field by the doctrine of nerve-centres, which is the most efficient fetish we have had yet. Is the gait affected? It is due to disease of the locomotor centres. Is there atrophy? The trophic centres are paralysed. Hypertrophy? The trophic centres are rampant. Glycosuria? The glycogenic centre is out of order. Ataxy? The coördinating centres (!!) have gone wrong. Have we a new and inexplicable symptom? Quick! invent a centre—two—three—half-a-dozen centres; destroy one, discharge another, and all is clear. For every motor disorder a separate centre is at fault; and when we study aphasia, we must first go back to Euclid and refresh our remembrance of geometry before we can hope to unravel the complicated diagrams in which the relative positions of the centres are demonstrated with such mathematical certainty.

Far be it from me to disparage the study of the localisation of functions in the nervous system. The discovery of this localisation is without doubt the most important event of this generation, and in the history of physiology ranks second only to that of the circulation of the blood. But while we admit, while we proclaim the localisation of function, let us remember to say that the localisation of representation is not absolute, but partial only. In the lowest centres of all the localisation is no doubt extreme. Such centres represent a limited part of the body very strongly; they represent little else, and that little but feebly. But in the highest regions each centre represents a large part of the organism preponderatingly, a still larger part in less degree, and the whole of the organism in some degree. And in the intermediate centres the representation is intermediate in character, a larger or smaller area being preponderatingly represented, and the halo of partial representation being larger or smaller, while the intensity of representation is less or more, according as the centre is more or less elevated in the hierarchy of the nervous system.

To the physician, and especially to the alienist, the establishment of the principle of the universal representativeness of the highest nervous centres is of profound importance. It clears up some of the obscurest and most recondite problems, both of medicine and of insanity. There is an old and familiar observation, that insanity has many physical accompaniments. The skin is altered; it sweats unduly, or is unduly dry, or desquamates more freely than normal. The hair is altered; it is harsh and staring. The nails get ridges on them, or are even discarded. The digestive function is altered; the whole process of nutrition is disturbed throughout the body. The lunatic is a lunatic, as Bucknill says, to the tips of his fingers, and, as Darwin says, to the end of every hair. And we now see why. We know that when a muscle is paralysed it quickly atrophies—when the skin is insensitive it readily sloughs; in other words, derangement of the function of a nerve-centre modifies not only the function of the part represented in the centre, but the nutrition of that part also. And hence derangement of the highest nervous centres disorders not only that adjustment of the organism to its environment, which it is the special function of these centres to effect, but disorders also the nutrition of every part represented in them —that is, the nutrition of the entire organism.

In this doctrine we find, moreover, the explanation of those almost miraculous bodily changes which occasionally follow upon tremendous storms of emotion. Such a change, for instance, is the rapid blanching of the hair, completed it may be in a single night, nay, in one recorded case within an hour (*vide* "Data of Alienism," 'Jour. Mental Science,' Oct. 1884.) Such changes are the pigmentation of the skin and other phenomena alluded to in the article above mentioned. Of course the principle here upheld does not show us the actual mechanism or chemistry of the changes in question, but that is a detail. Granted that a modification of nerve-currents modifies the nutrition of the parts into which they flow (and of this there is no longer any question), and the general alteration of nutrition throughout the body consequent on a commotion in the highest nervous centres becomes, on this principle, no longer a cause for wonder, but a necessity of natural causation. In the case of insanity, the alteration of general nutrition is mild and long enduring, in correspondence with the slight and chronic derangement of the highest nervous centres which is its cause. In the cases adduced in the article above-mentioned, the change of nutrition is sudden and astonishing in correspondence with the sudden and violent perturbation of nerve function on which it depends. Both cases are additional evidence as well as striking illustrations of the principle here advanced.

But what, it may be said, is the relevance of this discussion in an article on coma? How does it help to elucidate the nature of coma to show that the highest nervous centres in which coma begins, represent the entire organism? Why, in this way: that assimilates the subject of this article to other and widely different maladies, and shows in fact that *coma is a form of insanity.*

Let me not be understood to mean that every patient with incipient coma should be forthwith locked up in a lunatic asylum. The botanist who declares that an onion is a kind of lily, is not necessarily understood to mean that his bed of onions should be immediately transplanted into his flower garden. I speak not of insanity in the legal sense. But I wish to emphasise the doctrine that insanity is not a thing *sui generis,* and separated by an impassable gulf from all other maladies. If we want to unravel a tangled skein, we do not begin in the middle; we first find an end. And if we wish to comprehend insanity, we must not begin with the most striking and most histrionic cases, which first arrest attention, but patiently work backwards to the points, and they are many, at which the unique and inexplicable phenomena of insanity show their community of origin and of nature with those of other maladies, of which we have a better comprehension; and at these points we must begin our investigations. One such point of junction we have in coma, a disease which, from the preponderance and gravity of its bodily symptoms is included in the domain of the physician, but which is nevertheless in essence and in fact a form of insanity.

Coma is not merely a form of insanity, in the sense in which the delirium of fever and of belladonna poisoning are forms of insanity. It is much more. It is *the* form of insanity. That is to say, it affords in brief an example of the course that every case of insanity must go through. It is a very rapid insanity, it is true. Just as an epileptic fit exhibits all the movements of a fortnight or a month jammed up into a few seconds of time, so an ordinary case of coma displays in a few hours the events that in another vesania may be spread over many years. And whereas in the case of ordinary coma the course of events is commonly distinctly progressive or retrogressive, so that a few hours makes a decided difference in the aspect of the case; in that prolonged coma which we call insanity the progress of affairs may be arrested at almost any stage, and the case present little discernible alteration for years together. From one cloud a drop of water may fall into the sea in a few seconds. From another cloud at the same altitude, a drop may fall on the top of a mountain, may spend years in a snowdrift, centuries in a glacier, years again in a lake, and weeks in a river before reaching the level of the sea. But no one doubts that the cause of the descent was the same in the two cases. So it is with coma and insanity. Coma is a fulminant form of insanity; insanity is a lingering form of coma. Pathologically, coma is loss of function of the nervous centres, beginning in the highest centres of all; in those centres which are the substrata of consciousness, which effect the adjustment of the organism as a whole to its environment, which represent, first and most, the most precise and elaborate bodily movements, and which represent in some degree every part of the organism. From this commencement the loss of function spreads steadily downwards, to nervous regions successively less and less concerned with the manifestations of consciousness; to regions which effect adjustment of a more and more general, simple and fundamental character; to regions which represent more and more restricted portions of the organism. Or, translating this march of pathological events into its concomitant course of symptoms, there is first a dimness of consciousness, with a misconception of the relation of the organism and its surroundings, which we call being fuddled, with an inability to perform the most elaborate acts, and with an affection of some kind of the entire organism. From this commencement the progress is from dimness of consciousness to total loss of consciousness, from misconception of the organism to its surroundings to complete absence of such conception. From an inability to perform the most elaborate acts to an inability to perform the simplest acts, and then to an inability to perform simple movements; and from loss of complex and special movements to loss of simpler and more general movements, until at last nothing remains but the movements of the simplest parts of the breathing apparatus, and movements of the heart. Finally the breathing, due to compound tetanic muscular contraction, ceases, and nought remains but the isolated simple contraction of that fundamental morsel of the organism—the cardiac muscle.

Similarly, insanity is from a pathological standpoint a loss of function of the nervous centres, beginning in the highest of all, and often remaining restricted to these highest regions for years and years together. As in commencing coma, there is dimness of consciousness; as in that there is misconception of the relation of the organisms to its surroundings, which we now call delusion or illusion, or generally unsoundness of mind; as in coma, there is inability to perform certain acts of the more elaborate kind; and as in coma, there is some affection of every part of the organism. At this stage the insanity may remain. From this stage it may recover, precisely as the incipient coma of alcohol may recover if no more alcohol be taken. But suppose it do not recover. After weeks or months, or years, or almost a lifetime, one thing invariably happens. The teaching of experience is, that every case of insanity, if it goes on long enough, and is not cut short by some intercurrent malady, will invariably and inevitably at length *subside into dementia.* And what is dementia? What are its symptoms? In dementia the consciousness is dull; the vacant countenance, the hanging lip, the lack-lustre eye, the useless attitude of the hands, are all evidence that consciousness is defective. It is so defective, that if pinched or pricked the patient displays little or no concern, and he will let his toes char before the fire without moving his feet. Not only is consciousness extremely dull, but there is great lack of perception of the relation of the organism to its surroundings. The dement who has sat in the same chair every morning for months, and who has been taken from that chair to a particular place at a table half-a-dozen yards off three times a-day during the whole of that time, yet cannot, unaided, find his way from one customary seat to another. Every act of even a moderate degree of elaborateness is lost to him. He cannot even cut up his food. With difficulty and imperfectly can he carry it to his mouth. Not only this, but those movements that he can perform are uncertain and feeble. In other words, there is some paralysis. Together with this greatly defective consciousness, this profound lack of perception of the relation between himself and his surroundings, and this inability to perform acts of even moderate precision, there is a profound affection of the entire organism. The skin is sallow, earthy looking, sweaty or glazed; the hair is harsh; the bowels are constipated; the sphincters are not under control; the hands and feet are blue and swollen; the saliva is increased and altered. Compare these symptoms with those of a moderate degree of coma. They are marvellously similar. Allowing for differences due to difference in the rapidity of the malady, they are identical.

Finally, notice the termination of insanity. The majority of chronic cases are terminated by intercurrent maladies; but the normal and inevitable end of every case of insanity that does not recover, and is not cut short by a quasi-accident, is in coma—true, profound, unquestionable coma. Every case of acute delirious mania that ends fatally, ends in coma. Every case of dementia is liable at any moment to fall into coma, and many cases do so end. Even where they do not so terminate, it is common for dements to have little lapses into apparent unconsciousness, with still greater loss of power; and these attacks, which are manifestly of the nature of coma, are as manifestly exaggerations of the patient's usual condition of dementia.

Should it be objected that the over-action, the shouting, the restlessness, the furor, the eagerness of mania are fundamentally different and opposite in nature to the quietude, the prostration, the paralysis, of coma, I say that the comparison is wrongly made. The over-action of insanity must be compared, not with the stuporose stage of coma, with which it does not correspond, but with what I have called the precomatose stages of coma, with which it does correspond. Taking once more for illustration the coma due to alcohol, the parallelism between its stages and the stages of insanity, between its forms and the forms of insanity, is most striking, most unmistakable, nay, almost grotesque. Before the maniac subsides into dementia, he may be violent and ferocious; and before the drunkard subsides into somnolence, he may be quarrelsome and pugnacious; or the one may be melancholic, and the other maudlin; or the one have grandiose delusions, and the other be a braggart; or the one become lascivious, and the other amatory. It may be laid down categorically that there is no form of insanity whatever that may not have its counterpart in a case of drunkenness. How often have not the faulty articulation, the jovial aspect, and imperfect gait of general paralysis recalled to mind the early stages of drunkenness? The resemblance is not accidental, it is a fundamental similarity of nature, which of necessity exhibits itself upon the surface also.

Whether in origin, or in course, or in termination, coma and insanity are pathologically and symptomatically identical, and the only important difference between them is the difference in duration.

If this be so, then I maintain that we have made a considerable step forward, and gained a very important position from which to study both coma on the one hand, and insanity upon the other. The great duration of so many cases of the latter malady, tends to obscure their stages, and to obliterate from our minds their general course. We may know a patient for years, and know him only as a maniac, a melancholic, or a dement; but if we investigate his whole history, and see it to the end, we shall see that whatever form his insanity may most prominently and for the longest time display, in essence and in fact it is a case of coma. One stage may be enormously prolonged, and another evanescent or apparently absent; but the pathological processes, however various in duration, and however different in proximate nature, run in all cases the same ultimate course; they begin by ablating the function of the highest of all nervous regions, and they spread downwards as a mist rolls down a mountain, obliterating in succession the functions of the various nervous regions in their order from the highest to the lowest.

It will be remembered that the highest nervous processes are the latest and least organised, and that the lowest processes are the longest and most completely organised; so that the process which obliterates the nervous functions in their order from the highest to the lowest, is a reversal of the process of Evolution,—a disorganising of parts in the inverse order of their organisation. Hence, by taking the view of Coma that is here advocated, we are able not only to assimilate this malady to the superficially-different malady Insanity, but to bring both together as typical and regular examples of the operation of one fundamental law—the Law of Dissolution.

A CONTRIBUTION TO THE PATHOLOGY OF DREAMS AND OF HYSTERICAL PARALYSIS.

BY CH. FÉRÉ, M.D.,

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FALRET, Moreau de Tours, Brierre de Boismont, have spoken of hallucinations that begin during sleep and are reproduced for several nights consecutively, and end in being finally accepted as realities during day-time. Faure[1] and Richter (of Pankow)[2] have given cases in which dreams have been the starting-point of persistent diurnal deliriums. Suicidal or homicidal impulses have been observed after two or three nights of dreaming; occasionally a single dream is enough to set up a mental disturbance that manifests itself the next day.[3] Lasègue[4] has insisted upon the relations between dreaming and alcoholic delirium. I have had myself[5] opportunities of showing the analogy that exists between hysterical and alcoholic delirium in connection with the dreaming state.

Nor is it with delirium only that dreaming has intimate connections. Nothnagel and others have justly remarked that dreams may play an important part as a determining cause of epileptic attacks. The nocturnal fits occasionally observed in hysteria are as a rule caused by terrifying dreams, usually connected with the moral shock that determined the first seizure in the patient. In a case I saw at the Salpêtrière, the first hysterical attack was caused by a dream of alcoholic origin. The young woman, who had never before shown any signs of hysteria, was made to drink to excess one day by her brothers as a joke. She fell into a deep sleep, during which she had frightening dreams of fantastic animals going to devour her; without awakening, she entered a hysterical state that lasted two days, and followed by frequently repeated attacks. I wish in this paper to call the reader's attention to a peculiar accident, resulting from a terrifying dream in a hysterical patient.

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[1] Faure, "Etude sur les Rêves morbides;" ('Arch. Gén. de Méd., May, 1876.')

[2] Richter, 'Arch. f. Psychiatrie,' xiii. 1.

[3] R. Ball, "Des Rêves prolongés" ('France Médicale,' 1886).

[4] Lasègue, "Le Délire alcoolique n'est pas un délire, mais un rêve" ('Arch. Gén. de Méd.' 1881).

[5] Féré, "La Médecine d'imagination" ('Progrès Médical,' 1886, No. 37).

Hysterical Paralysis after a Dream.—Aphasia with Paragraphia. Blepharospasm, &c.

Eugénie P. came to the out-patients' room at the Salpêtrière on the 11th of September, 1886. Her mother, aged 40, is corpulent and quick-tempered, though she denies having ever shown any nervous symptoms; a minute cross-examination reveals nothing further. The father is not related to his wife, but is of nervous temperament, irritable, but with no marked neurotic history. He is in the wine trade, and his sobriety is problematical. No full account of the collaterals can be obtained. There have been three children; the eldest, aged 18, is a girl in very good health. The next is our patient; the third was a boy, who died of meningitis at 18 months.

E. P. is 14 years of age. She came to term and spoke and walked at about 14 months; was clean early, and had no convulsions nor any other early nervous symptoms. She suffered from so-called "hemicrania" as early as between her sixth and tenth year; these attacks were violent enough to suggest meningitis, and she seems to have had delirium after several short feverish ailments. She also was subject to nightmare and nocturnal terrors. The catamenia appeared last January, and are said to have been regular till July. The period of August was missed without any concomitant symptom, though since that time E. P. has had more restless nights; she dreams more, and is rapidly growing taller. In the night of the 20th to the 21st of August, E. P. dreams that she is pursued in the Place de l'Odéon by men who want to stab her; she runs with all her might, and at last escapes them. She awakes exhausted and bathed with perspiration. The next day her mother noticed while walking out with her that the child's legs were giving way at intervals, though she did not complain of fatigue. The dream occurred again the following night; and the next day, Sunday, the phenomena observed before were still present; in addition to these the child was inattentive, not answering questions as usual; at times during the day she appears frightened, and says that it is her dream which is coming on again.

The same dream renews itself during the following night; the patient is again awakened in the same way, perspiring and exhausted. The weakness of the legs becomes more marked, she falls several times in the day. Day dreaming becomes more marked; the terror movements are more frequent and intense. She sees men pursuing her, and hears them calling out to her; she feels them seizing her hair. At times she breaks or drops some object she is holding in her hand, in order to prevent their taking it away.

The parents make her take walks as a means of distraction, and improvement of the sleep. But they find that this going out becomes impossible on account of the patient, who does not complain of any fatigue in her legs, giving way and collapsing to the ground.

This condition of diurnal and nocturnal dreaming persisted without any change till the 7th of September, when it diminished in intensity. E. was no longer terrified during the day, was calm and enjoyed sleep in the night of the 9th. The weakness of the legs appears, however, to be on the increase. During the morning of the 10th her sister calls her to try on a dress on the floor above. She rushes upstairs, but on reaching the top her legs cease to obey, and she sinks helpless to the ground. From this moment the limbs remain flaccid till the evening, when she begins to make a few inco-ordinated movements. She moreover appeared to be unconscious of her condition. She had no dreams during the next night either.

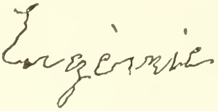
On the morning of the 11th the patient is brought to the Salpêtrière, two persons carrying her under the arms. E. P. is rather tall for her age, with a dark, dull complexion, giving one at first the impression of a good constitution.

When at rest the physiognomy appears regular, but on closer inspection it is noticed that when she laughs the mouth deviates a little to the left; moreover when at rest, the naso-labial fold is more marked on that side. It seems as if the face be a little smaller on the same side. The two irides are of the same colour, but the pupil on the right is somewhat smaller. There is some ovarian tenderness on the right, as well as hemianæsthesia, slight in the arm, very marked in the leg. The special senses are likewise involved: she hears the watch at a less distance, taste is less developed, smell and sight less acute; the visual field, likewise, measured by a rough test, seems to be a little contracted on the same side. She does not feel so well on the left leg as on the left arm. E. is incapable of standing, and collapses as soon as the support is withdrawn. The knee-jerks are exaggerated, especially on the right. When she sits, the toes hang downwards. The tendon-reactions at the right wrist are lively, whilst the dynamometer shows 18 on that side and 24 on the left.

When, being supported, she is made to perform the movement of walking, the foot is seen to advance; but when the heel is near touching the ground, a sudden spasm of the calf occurs that causes the toes to strike the floor, and the heel comes down an instant afterwards. This kind of "dicrotism" is noticed at every step. I prescribe a tonic regimen, three grammes of bromide at night, as well as rest and two daily sittings of passive movements of ten minutes each. These passive movements are carried out while the patient is reading aloud, so as to avoid the resistance of the gastrocnemii, which still are influenced by the will. On the 15th of September E. is seized with a fit of choking and crying, loss of consciousness, and a few spasms, followed with abundant micturition and defecation. On the 20th she comes again to the consultation. She has had no more dreams, and has recovered her usual temper. She walks on the arm of her mother, though the legs are still very weak, and the dicrotism still persists. The same treatment is continued, with cold douches to the trunk and limbs for half a minute every morning at the same time. On the 27th the improvement is still more marked. E. can walk by herself in the room, but the movements of the hips show that the trunk muscles are needed to supplement the yet paretic muscles of the legs; the patient has thus a dancing gait. Same treatment. Moreover, walking exercises to command, twice a day for fifteen minutes. On the 4th of October E. came on foot, without assistance, from the station to the hospital, a distance of about 300 metres. For some days past she goes out a little, walks by herself, though still with the same gait.

On the 9th, after her douche, her mother wanting to go out undresses her and puts her in bed, so as to prevent her from going about during her absence. E. enters into a fearful passion, shouts and talks wildly for two hours; after which she falls into a deep sleep. When her mother wakes her up on returning at midday, E. cannot say one word. She understands what she hears, moves her tongue properly, but no sound issues from her mouth. They try to make her write, but she cannot, though the movements of the fingers are perfect; she tries to trace the form of the letters, but is unable to make a single one regularly. In the course of the evening she begins to articulate a few sounds. On the 11th of

October, when she comes to the consultation, the patient talks in a jerky way and misses syllables; her tongue is the seat of choreiform movements. Though able to do fine needlework, she has the greatest difficulty in writing even her own name; she hesitates before beginning to write a letter and traces it tremulously. She walks much better, is able to run; the peculiar movement of the hips is still noticeable, but the dicrotism is present only on the right side, which is weaker, and has more lively tendon-reactions. *October* 18*th.*—The catamenia have lasted three days, and were as abundant as in the past. Speech and writing have returned, the gait is much more normal. *Oct.* 25*th.*—Walking still further improved; the right leg only gives way a little after a long walk. Yesterday, however, after a fright caused by the fall of a piece of furniture, another symptom was developed, viz. a spasm of the eyelids, which move sometimes as often as 100 times in a minute. This spasm is accompanied with a convulsion of the superior rectus which remains contracted for fifteen or thirty seconds at a time; the eyeball is drawn completely upwards, so that vision is impossible. This spasm disappears within two days.



Eugénie

I saw the patient again on the 18th of November, when every trace of the symptom had disappeared except some right hemianæsthesia.

This case of paralysis after a dream deserves to fix our attention. It enables us, I think, to give another explanation of the origin of the so-called psychical paralyses as described by Profs. Russell Reynolds, and Charcot. It is assumed that these paralyses arise from an idea or suggestion; in other words, that the motor trouble comes on after its mental representation. This theory has even been applied to paralyses from traumatic causes. The circumstances under which the symptom has appeared in my patient, seem to me to support the theory of paralysis through exhaustion which I have propounded for traumatic paralyses.[1] The weakness of the limbs is really produced by the gradual influence of fatigue due to the exhaustion of the motor centres through a rapid succession of discharges ineffectual to produce movements. This same patient has moreover offered us another example of a fatigue paralysis, when she became incapable of phonating after the discharge of another centre.

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[1] 'Bull. Société de Biologie,' 1886.

I may add that, by calling forth dreams of running in hypnotisable subjects during their normal sleep, I have been able to produce similar paralyses that were accompanied by the same "dicrotic" gait, which we saw was due to the preponderating action of the flexion. In a certain number of cases of so-called psychical paralysis, the symptom supervenes after protracted cerebral work, and not after a subjective representation of fatigue or loss of power. Let us notice also in this case the disturbance in the writing which is an exceptional symptom in the history of hysterical asphasia. But the chief object of this paper is to draw attention to the influence of dreams in the development of certain psychical troubles. Our observations tend to show that dreaming, and especially repeated dreaming, must not be considered as an indifferent phenomenon, but often constitutes the opening scene of a morbid drama, and as such deserves the attention of the physician.

ON CHANGES IN NERVOUS SYSTEM AFTER AMPUTATION OF LIMBS, WITH BIBLIOGRAPHY AND RECENT CASE.

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The changes which occur in the nervous system after the amputation of a limb, and which are probably due to the disuse of the member, are both interesting and instructive, as giving some slight clue to the functions of various nervous elements. Though an apparently trivial subject, still its literature is both full and fairly exhaustive, and this paper is intended rather to epitomise the results obtained up to the present, than to furnish many new facts. The changes noted in the peripheral nerves and cord, and published before 1880, were collected together by Dreschfeld in a paper entitled "On Changes in the Spinal Cord after Amputation of Limbs."[1] From this I have obtained the earlier references, looking up such of the cases as I had access to.

History.

Bérard, in 1829, found atrophy of the anterior roots supplying an arm which had been amputated. *Vulpian*, in 1868, reported two cases; one in which amputation was performed forty-seven years before death, where the cord was lessened in size both in grey and white matter on the amputated side, but without alteration of the cells; and a second (amputation twenty years ante mortem) with atrophy of the anterior white and grey matter, and of the motor ganglion cells, together with granular degeneration in the anterior grey matter. In 1869 he again reported two cases with changes in the posterior white and grey matter; also a third case (amputation a year and a-half ante mortem) with changes in the anterior grey and white matter, but none posteriorly. Experimenting on animals, he found that tearing or irritating peripheral nerves, caused ascending degeneration in the cord. *Hayem* (1873) obtained the same results, but *Cruveilhier* and *Türck's* experiments were negative.

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[1] 'Journ. Anat. and Physiol.' 1880, xiv. p. 424.

Dickinson reported three cases in 1869. The amputations were all of more than twenty years' standing. In one there was a neuroma at the cut end of the sciatic. This showed a granular appearance from the presence of minute circles (with no difference of grey or white matter), separated by structureless material giving a very pink section after staining; there was much connective tissue round, but both sciatics were the same to the naked eye. The changes found in the cord were, diminution of the grey matter of the posterior column (especially the anterior extremity), wasting of the nerve roots, especially the posterior; all being found on the side of the amputation. There was found no change in the cells. He points out that Waller's theory of degeneration does not hold in these cases. His diagrams only show relations of size. *Lockhart Clarke* examined some of the last-mentioned cases, and found atrophy of the postero-lateral cell group of the anterior horn. *Friedreich* found the cord normal after an amputation of twelve years' standing. *Erlenmeyer* (1872) found the nerve fibres on the amputated side smaller than in the opposite limb. *Dickson* (1873) noticed atrophy and diminution in number of the cells of the anterior horn, especially in the postero-lateral group in a fifteen-year-old amputation of the leg. *Genzmer* found atrophy of the large cells of the anterior horns in the lumbar region (amputation of right thigh thirty-three years ante mortem). His plate shows also marked atrophy of all parts of the right side of the cord. *Leyden* gives three cases, all with corresponding atrophy of the cord, especially in the external part of the grey matter. *Dreschfeld* (1880) reported a case (amputation of left thigh fifteen years ante mortem) showing an amputation neuroma. There was no change in the sciatic, nor in the anterior or posterior roots; the brain was normal. The left side of the lumbar cord showed few cells, and these were atrophied but not pigmented; they had no branches, and many no nucleus. At the external lateral border of the anterior horn, there was granular material with fibrils surrounding the cells, and staining well. Higher up, the groups had the full number of cells, excepting the intermedio-lateral group, which was still atrophic.

Hayem (1876) found fine fibres in the nerve stump grouped into bundles. He concluded that, in the place of degenerated nerve-bundles, whole bundles of fine nerve-fibres are built up. He found the new fibres larger in a twenty-four-year-old amputation than in one five years old. *Déjérine* and *Mayor* (1878) found an atrophy of nerve fibres in the cut nerve. This atrophy decreased upwards towards the cord, and disappeared completely at a certain distance above the amputation level. In the cord they found atrophy of grey and white substance in all parts on one side, without change of the nerve fibres and without decrease of the ganglion cells generally. But in one case of thigh amputation there was in the mid-lumbar region a diminution of cells to the extent of one-third, especially in the inner anterior group of the anterior horn. They found no microscopic changes in the nerve roots. *Erlitzky* amputated the limbs of young dogs and found lessening of the posterior root, of the posterior columns and posterior horn, with diminution in number and size of some cells of the anterior horn of the same side. *Edinger* noticed atrophy of the whole half of the spinal cord in a case of intra-uterine amputation of the hand, but the ganglion cells were unaffected. Later, he found in two cases of amputation only a slight atrophy in the lumbar cord.

Hale White and *Edmunds* in 1882 showed amputation neuromata to the Pathological Society. They found that the first change in the coiled bundles of nerve fibres at the end of the nerve was an ingrowth of delicate connective tissue from the perineurium. This caused sclerosis of the bundles, which then underwent fatty degeneration. Some of the fibres remained healthy, these being probably connected with parts of the limb still remaining. In one case (amputation of thigh) there were changes in the tractus intermedio-lateralis in the lumbar region only.

Hayem and *Gilbert* (1884) publish a case of amputation of the forearm just below the elbow forty-seven years ante mortem. The brain was normal. There were neuromata on the ulnar, median and radial nerves in the stump. These were composed of a large number of small nerve-fibres, formed of an axis-cylinder covered with a thin layer of myeline and grouped in fasciculi. They were much intertwined, but for the most part the direction was the same as the fibres of the main nerve-trunk till they approached the periphery of the neuroma, where they were looped back. The relation to the healthy fibres seemed to be, that at different levels the healthy fibres perished and were each replaced by a number of small thin nerve fibres, but the actual point of continuity between the large and small fibres was not observed. The neuromata appeared therefore to be due to a regenerative process, new nerve-fibres growing from the cut end of the nerve. The affected radial was of diminished size, but the median remained the normal size. In both, however, the nerve fibres were atrophied, and there was a growth of interstitial connective tissue. The ulnar was increased in thickness and here were found two forms of nerve fibres: (*a*) large isolated fibres with a fine axis-cylinder and thick coating of myeline, accompanied by a longitudinal band of connective tissue; (*b*) fasciculi of small fibres with a comparatively large axis-cylinder and thin coating of myeline; the interstitial connective tissue of the nerve was increased. The anterior and posterior roots of the brachial plexus were atrophied, and contained more small fibres than normal. In the cord the posterior columns of the affected side were smaller, but for the other columns the reverse was true. There was atrophy of the grey substance (particularly at the level of 7th and 8th cervical and 1st dorsal nerves), affecting chiefly the cells of the anterior horns.

Dudley gives a case of amputation of the left leg of a general paralytic twenty-one years ante mortem. I have to thank him for his great kindness in allowing me to see his specimens. The brain was fairly symmetrical, excepting that the right ascending frontal convolution was somewhat smaller than the left. He is the only one to note the microscopic appearance of the cortex in these cases, but he found no change which could not be accounted for by the general paralysis. The cord was healthy in the cervical and dorsal regions. In the lumbar region the left anterior cornu was much reduced in size, and the cells were less numerous and smaller, especially in the antero-lateral and postero-lateral groups. Many cells were pigmented. The left posterior cornu was slightly reduced. The left anterior nerve-roots within the cord were reduced in number, and in some the axis-cylinder was broken up and granular. The left anterior crural nerve was the only one examined. It showed very fine fibres, scattered irregularly between healthy fibres in groups which stained deeply; the diminished size seemed principally due to the attenuation of the medullary sheath. There was much adipose tissue between the fasciculi.

Just as I was finishing my own investigations, Dr. A. de Watteville kindly forwarded me 'Fortschritte der Medicin,' No. 23, Dec. 1st, 1886. This contained an important paper by *C. Friedländer* and *F. Krause* on the changes in the nerves and spinal cord after amputations. They give a bibliography of seventeen references, five of which I had not hitherto known of, and which I have added to my bibliography. It is somewhat curious that my method resembled theirs in almost every detail; but I shall have occasion to criticise their conclusions. They investigated eight cases (of from 3 months to 10 or more years' standing). They found a degeneration of the cut nerves quite different from the Wallerian degeneration. The peripheral nerves showed normal bundles broken up by atrophied bundles; the latter were very small, and separated by much wider spaces. They contained atrophied fibres with faint outlines and completely lustreless, occasionally a normal glistening medullated fibre being seen amongst them. The atrophied fibres did not give a blue coloration with Weigert's copper hæmatoxylin stain, but only a yellow colour, showing the absence of myeline. With aniline-blue and ammonia-carmine, the smallest circles showed a central point which was probably the remains of an axis-cylinder, but this was not noticed in longitudinal section. There was in the atrophied bundle a considerable increase of nuclei. No new fibres were recognized, the atrophied fibres appearing equal in number to the formerly existing medullated fibres. These changes were found 3 months after amputation, but the fibres were not so much lessened in size as in cases some years subsequent to amputation. As the cord was approached, the number of normal fibres increased, these of course coming from the parts still remaining. They examined both anterior and posterior roots (central to the ganglion on the latter), and found no change, except perhaps a slight diminution in the number of fibres of the posterior root. The spinal ganglion contained many atrophied fibres. The piece of sensory nerve, however, between the ganglion and the junction with the anterior root was almost entirely atrophied, only a few normal fibres remaining. In the cord they found a decrease of the posterior column, also slightly of the posterior horn, but no affection of the nerve fibres. There were no changes in the antero-median, antero-lateral or central groups, but marked decrease in number of the cells of the postero-lateral group and of the vesicular column of Clarke. The differences in the latter, however, were slight as compared with the former (5·3 : 7 in Clarke's col., 1 : 2·5 in the postero-lateral group). The total decrease in size of half the cord was entirely due to the diminution of the posterior column and horn and of the postero-lateral group. They conclude that the changes only affect the sensory sphere, that only those sensory fibres connected with touch-corpuscles and end-bulbs are atrophied (a case of amputation near the ankle showing as many atrophied fibres as one through the thigh), and finally that the cells of Clarke's column and of the postero-lateral group are sensory in function. They promise in another paper to give their explanation of the atrophy of the fibres. I have quoted this paper somewhat fully, as the conclusions are so much at variance with generally accepted opinions.

The remaining cases relate only to the changes which have been observed in the brain. *Féré* and *Mayor* (1877) describe the brain of a man whose leg had been amputated thirty years before death. In 1878, *Ch. Féré* published a paper entitled "Note sur les Cerveaux d'amputés," and *A. Mossé* one entitled "Note sur un Cerveau d'amputé." Unfortunately I have not been able to see any of these papers, so cannot give particulars. *Oudin* (1878) gave a case of a woman who had an injury to the right leg sixty-six years before death; it at once became useless and remained atrophied, and she spent the rest of her life in the sitting position. After death the brain was found to be asymmetrical, the left first frontal convolution was narrowed posteriorly, nearly rectilinear and without sinuosities; the anterior part of the left superior parietal lobule was also atrophied, and the convolutions were small. The ascending parietal convolutions were narrow, but more so on the left side; the left fissure of Rolando was shorter and more anterior than the right. No other changes were noticed either in the brain or cord. He adds that such an atrophy of the brain would probably result only if the limb were rendered useless in infancy. *Charcot* (1878) showed a photograph (to a Society at Paris ?) of a brain thirty-eight years after exarticulation of the shoulder. There was no difference on the two sides In this case *Déjérine* and *Raymond* found in the cortical region of the cord on the right side, a sclerotic atrophy of the lateral bundle, not reaching the medulla (was this Gower's tract ?). *Féré* had a few days before this communicated a similar case to the Société de Biologie. *Luys* mentions a case of amputation of thirty-five years' standing, causing atrophy of the brain. *Sanders* (cited by Charcot in "Les leçons sur les localisations") gives cases of brain atrophy associated with infantile paralysis. *Colard* and *Landouzy* give similar cases. In these, however, the question arises whether the brain atrophy was due to the disuse of a limb, or whether the cases were not rather spastic hemiplegia of infancy, the brain lesion being primary. *Pitres* in 1877 contributed an article to the 'Gazette médicale de Paris,' on partial atrophies of cerebral convolutions, consecutive to old amputations of limbs on the opposite side. *Varigny* (1879) has a note on a case of amputation as pointing to the localisation of cerebral function. *Bourdon* gives a case in which the left arm had been amputated forty years before death, this being followed by progressive paralysis of the left leg. He found an extensive atrophy of the motor zone of the right hemisphere, with a diverticular enlargement of the lateral ventricle towards the atrophic spot. *Gowers* (1878) reports a case of congenital absence of the left hand. He found a marked difference in width of the right and left ascending parietal convolutions about the middle third, the diameter of section of the former being ·35 in., and of the latter ·65 in., no other brain abnormality being noticed. This I mention as being somewhat allied to amputation cases, though of course the relation of cause and effect is here not so clear.

Recent Case.

I have recently examined the changes in the nervous system of a patient who died in the West Riding Asylum, Wakefield.

George W——, admitted Jan. 16th, 1875. æt. 35, was a labourer. He had been temperate, but had been insane for twelve months before admission. His mental symptoms were somewhat peculiar, but he was almost certainly an anomalous case of general paralysis with long duration, dementia being the chief feature. The left thigh was amputated through the lower third on Nov. 14th, 1879, in consequence of caries of the bones round the knee-joint. The wound was slow in healing, but no mention is made of him having suffered any special pain in the stump. He gradually became more demented, and died from phthisis April 2nd, 1886, 6½ years after the amputation. At the autopsy there were found thickened cerebral meninges, and excess of cerebro-spinal fluid, but there were no adhesions. The cerebral convolutions were fairly complex, but atrophied; there was no marked difference on the two sides. Pons and medulla appeared healthy, but were unfortunately not reserved for further examination. The lungs showed tuberculous pneumonia.

Special Examination.—The spinal cord macroscopically showed no changes in the cervical, dorsal, or upper lumbar regions. In the middle and lower lumbar regions, however, the cut section of the left half was much smaller than that of the right half. Both white and grey matter, but especially the latter, appeared to be affected; the left anterior nerve-roots were smaller than the right, but no difference was noticed in the posterior roots. Microscopically, the only changes seen in the cervical, dorsal, and upper lumbar regions were pigmentary degeneration of a few cells in the anterior cornua, and a slight increase of connective tissue in the lateral and posterior columns, both changes being easily accounted for by the general paralysis. No unilateral changes were seen. The cells of Clarke's column were carefully examined, and found very unequal in number on the two sides in any one section, but the decrease was about as often on the right as on the left side, so that it could not be due to the amputation. To settle this important point, I examined this region in other cords, and I found the same great difference in number in any one section, some sections showing more cells on the right, others more on the left side. In fact, as these cells probably are connected with viscera, there does not seem to be the same necessity for them to be equal in number in any plane, as if they supplied two similar limbs.

In the mid-lumbar region the left half was smaller in all parts, excepting the anterior column. The lateral column was slightly decreased, the posterior more so, and especially at its anterior end; consequently the posterior commissure was inclined forwards from left to right. Both the anterior and posterior cornua were smaller in all diameters, the nerve cells of the latter having fewer processes. The nerve cells of the left anterior cornu stained only faintly, were rounded and possessed few processes; they were mostly loaded with yellow pigment. With a high power faint outlines of much degenerated cells were seen. The neuroglia was more granular and more deeply stained. The cells of the right anterior cornu were normal, excepting that a few were slightly pigmented. The qualitative changes of the cells on the left side were accompanied by great quantitative changes. Six sections were taken indiscriminately, and the cells of each group counted, the following remarkable results being obtained:

No. of Section.	Left.						Right.					
	1	2	3	4	5	6	1	2	3	4	5	6
Antero-Intern. Group	13	11	8	14	7	6	22	19	7	10	8	5
Antero-Lateral ,,	6	7	7	6	11	8	9	17	8	13	15	6
Central ,,	6	6	8	6	4	8	7	17	19	9	6	13
Postero-Lateral ,,	8	7	4	4	4	4	21	12	24	23	10	22
	33	31	27	30	26	26	59	65	58	55	39	46
Total . . .	173						322					
Average . .	28·8						53·6					

All the above groups are seen to contain less cells on the left side, and this is especially the case with the postero-lateral group, and after this with the central group.

The filum terminale showed the left cornu (especially the posterior) to be smaller, but the posterior columns were much the same size.

The Nerves.—The sciatic and anterior crural nerves on each side were examined. Both sciatic nerves were of about the same consistency, but the area of section left was slightly larger than that of the right. This seemed to be due rather to an increase of the bundles themselves, which had a more fatty and less solid appearance on the left side. The same applies, in a much less marked degree, to the anterior crural nerves. At the end of the left sciatic was a small nodular mass (about $\frac{1}{2}$ in. × $\frac{3}{8}$ in.) covered with connective tissue; this was evidently an amputation neuroma. A much smaller one was found at the end of the anterior crural. The sciatic neuroma was cut longitudinally and transversely, and examined microscopically. It was surrounded by a thick bundle of connective tissue, which sent strong processes into the mass and separated the bundles one from another. These bundles were cut across in every imaginable direction, but superiorly were principally directed longitudinally. They consisted of closely aggregated very small nerve-fibres, composed in every case of a fine axis-cylinder surrounded with a thin layer of myeline. The fibres varied somewhat in diameter, the largest being about twice the size of the smallest, but only about one-sixth the diameter of a normal fibre (not one of which was to be found). What we may call the large primary nerve-bundles were widely separated by channels containing connective-tissue fibres and nuclei.

Each primary bundle was divided into several secondary bundles, separated by narrower channels. All these channels seemed to be connected, and were probably lymph spaces.

The left sciatic nerve was examined at various points. A section two inches above the neuroma (see Fig. 1) showed, with a low power, a slight increase, both in extent and compactness, of the connective tissue and fat between the bundles. The vessels showed no special change, and the perineurium was normal. In the bundle the lymph paths were wider than normal, separating the bundle into well-marked secondary bundles. The channels contained shreds of connective tissue and nucleated cells. The secondary bundles were also divided by narrower channels into minute groups. Some of these consisted of one or more fairly normal fibres with a little connective tissue around; from the great preponderance of myeline and a somewhat atrophied axis-cylinder they appeared lightly stained. Other groups consisted of a single normal fibre with a darkly-stained mass, and others again (and these very largely predominated) consisted only of a dark mass apparently containing no nerve fibres. Thus a primary bundle appeared to be made up of dark groups with an occasional light group, all being separated by wide channels. With a high power the dark masses were easily seen to consist of very minute medullated fibres, exactly like those in the neuroma. They were separated by a deeply-stained tissue, probably connective in nature. To test whether the fine glistening ring round the axis-cylinder was myeline or not, Weigert's hæmatoxylin stain for myeline was used, when, with very thin sections and good daylight, the blue-stained ring with the yellow axis-cylinder and connective tissue were beautifully shown in each case. On staining other sections with a watery solution of logwood many nuclei were discovered, principally between the minute fibres; most of these undoubtedly belonged to connective-tissue cells, but some may possibly have been nerve nuclei. I estimated after much care that the

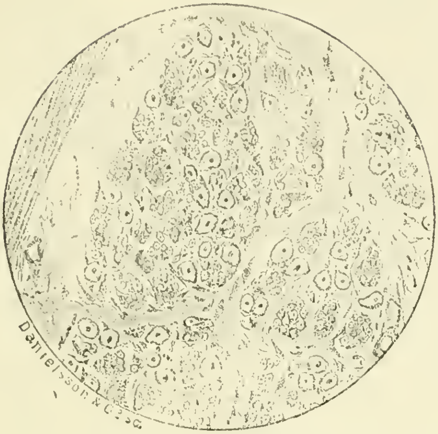

Fig. 1.—Transverse section of Left Sciatic Nerve in case of Amputation of Left Thigh, showing numerous small fibres and few large fibres. The spaces between the bundles should be quite clear, and not shaded as in the figures Both are magnified 300 diam. E. S. Reynolds, del.

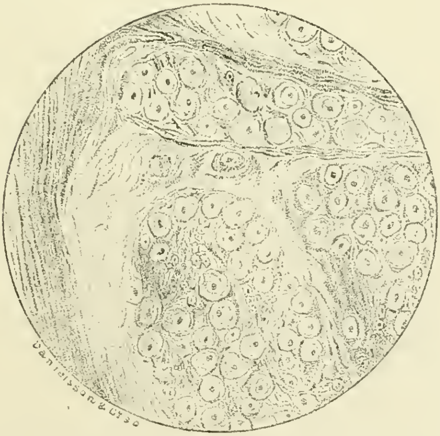

Fig. 2.—Transverse section of Right Sciatic Nerve in case of Amputation of left Thigh, showing numerous large fibres and few small fibres.

total number of fibres large and small, in one field, was about equal to the number seen in a normal nerve in the same field; it was certainly not less, and could not safely be said to be more.

At a point four inches above the neuroma much the same appearances were seen, but already the number of large fibres was slightly increased. Sections taken at the upper end of the left sciatic showed two distinct forms of primary bundles; a less numerous kind composed almost entirely of large normal fibres with a few patches of small fibres; and a much more numerous kind, just like the section at the end of the nerve, with patches of fine fibres (with a few large ones) separated by wide channels. Thus the only difference between the upper and lower ends is the addition in the former of a number of normal fibres, these being almost certainly distributed to the thigh.

The changes in the left anterior crural nerve were much the same as in the sciatic, but were found in a much less marked degree. This we should expect, as the anterior crural is so largely distributed to the thigh.

The right sciatic (see Fig. 2, p. 505) and anterior crural nerves consisted almost entirely of primary bundles of normal fibres. A few patches of small fibres were, however, found, but in very slight amount as compared with the left side.

The nerve roots on both sides were cut away from the cord, thus being, in the case of the posterior, for central to the spinal ganglia. The right anterior root showed nerve fibres beautifully uniform in size and closely packed together, not a single small fibre being found. The left anterior showed a large predominance of normal fibres, but they were separated into small groups by wide channels and increased connective tissue. In addition, the important fact was noticed that many small medullated fibres were seen mixed up in certain bundles with the large fibres. The right posterior root showed fibres varying in size from large to very small, but the former largely predominated; all were closely packed together. The posterior root showed the most marked changes. The fibres varied much in size, but the small predominated greatly over the large; they were divided into small groups by wide channels and connective tissue, forming a marked contrast to the closely packed fibres of the roots on the right side.

The left posterior spinal ganglia contained a large number of small fibres, and an increased amount of connective tissue, as compared with the right posterior spinal ganglia.

CONCLUSIONS.

The few small fibres of the right sciatic nerve are probably due to the general paralysis. This lesion was described by Dr. Bevan Lewis, in the 'West Riding Reports' for 1875, and he, from a theoretical point of view, considered them to be afferent; this seems to be the case, as I only found them in the posterior nerve-root.

The neuroma was seen to be made up wholly of small fibres. These I consider to be regenerative fibres, growing from the fibres above, and so lucidly described and figured by Ranvier, as occurring at the end of the central stump of a cut nerve.

The numerous small fibres of the sciatic trunk I look upon differently, however. They seem to be fibres simply atrophied in all their constituent parts, without any degeneration. This is due purely to disuse, the connection with the trophic centres preventing any degenerative change. Most of them are undoubtedly sensory, and the change could be traced distinctly through the posterior spinal ganglia right up to the cord. But very many are most certainly motor, as proved by the number of small fibres in the left anterior roots. Theoretically, we can easily see that after amputation, say of the thigh, no afferent impulses from the part removed can travel to the cord, and therefore these fibres are absolutely unused. But it is quite possible for motor impulses from the cord to overflow into the efferent nerves of the stump, even if most of them become blocked at the site of amputation. I only throw this out as a doubtful explanation of the greater number of atrophied sensory fibres.

In my case, as above stated, there was ample proof, from an examination of the nerves and their roots, that both motor and sensory spheres were affected. This being allowed, there is no reason to say that the postero-lateral group in the anterior horn is sensory in function. It was, moreover, by no means the only group of the anterior horn affected, as the central and antero-lateral groups were both markedly changed, again pointing to an implication of the motor sphere. The idea that the postero-lateral group is sensory is quite contrary to all former anatomical and pathological observations; its function almost certainly being, as pointed out by Ross, to innervate muscles which maintain the erect posture.

The affection of the sensory tracts was accompanied by a diminution in size of the posterior column and horn of the same side. It was curious that no unilateral ascending degeneration was noticed in any part of the cord. In my case no definite lesion of Clarke's columns was noticed, and remembering how the number of cells on each side in any one plane vary in this group, there seems hardly sufficient evidence, from the comparatively slight difference on the two sides in Friedländer and Krause's cases, to assert that it is sensory in function. It must, moreover, be remembered that Ross long ago suggested that the cells of Clarke's column were specially connected with the innervation of the viscera. This idea has been worked out and fairly established by Gaskell,[1] and adopted by Ferrier in his new work.

I thus finally conclude that, after amputations of limbs, the changes which occur may be explained simply, and might have been expected from à priori reasoning. The nerve fibres up to and with their centres in the cord atrophy from pure disuse. Each small fibre in the nerves has arisen from one large fibre, and the shrinking which thus would occur appears to be compensated for by the great widening of the lymph channels, and the slight increase of connective tissue in the small bundles.

The unilateral changes in the brain seem only to occur if the amputation is performed early in life.

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[1] 'Journal of Physiology,' vol. vii. No. 1.

BIBLIOGRAPHY.

(1) Bérard: Case shown before Anat. Soc. de Paris, 1829.

(2) Vulpian: 'Archiv. de Physiol.' 1868, p. 443; 1869, pp. 683, 687, 690. 'Comptes rend. de l'Académ. des Sciences,' 1872, p. 624.

(3) Hayem: 'Arch. de Physiol.' 1873, p. 504. 'Progrès médical,' iv. 1876. pp. 199, 518. 'Bullet. de la Soc. anat. de Paris,' 1875, Series III. t. x. p. 634; and 1876, Series IV. t. i. p. 230.

(4) Erlenmeyer, Albrecht, jun.: 'Ueber das cicatricielle Neurom. Greifswald,' 1872, p. 383.

(5) Dickinson: 'Journ. Anat. and Physiology,' 1869, iii. p. 88, with drawings.

(6) Lockhart Clarke: 'Medico-Chirurg. Transact.' li. p. 249, with plate.

(7) Friedreich: 'Progress. Muskelatrophie,' p. 14.

(8) Dickson: 'Patholog. Transact.' xxiv. 1873, p. 2.

(9) Genzmer: Virchow's 'Archiv,' lxvi. with plate.

(10) Leyden: 'Klinik der Rückenmark-Krankheiten,' vol. ii. p. 316.

(11) Dreschfeld: 'Journ. Anatomy and Physiol.' 1880, xiv. p. 424.

(12) Déjérine and Mayor: 'Comptes rend. et Mém. de la Soc. de Biol. Paris, 1878, xxx. 'Progrès médical,' 1878, p. 600.

(13) Erlitzky: 'Petersb. med. Woch.' 1880, pp. 38, 45.

(14) Edinger: Virch. 'Archiv,' Bd. 89, p. 46.

(15) Hale White and Edmunds: 'Lancet,' 1882, ii. p. 113.

(16) Hayem and Gilbert: 'Archiv. de Physiol.' May 1884, p. 30. Abstract in 'Brain,' 1885, No. XXX. p. 283, by J. A. Ormerod.

(17) Dudley: 'Brain,' 1886, No. XXXIII. p. 87.

(18) C. Friedländer and F. Krause: "Ueber Veränderungen der Nerven und der Rückenmark nach Amputationen." — 'Fortschritte der Medicin,' Dec. 1, 1886.

The following refer only to the state of the brain :—

(19) Féré et Mayor: "Cerveau d'un homme amputé de la jambe depuis trente ans."—'Bull. de la Société anat. de Paris,' 1877.

(20) Féré, Ch.: "Note sur les Cerveaux d'amputés."—'Gazette médicale de Paris,' 1878, No. 3.

(21) Mossé, A.: "Note sur un Cerveau d'amputé."—'Bullet. de la Soc. anat. de Paris,' 1878.

(22) Oudin: 'Revue mensuelle,' 1878.

(23) Charcot: Case communicated to Paris Society, 1878.

(24) Féré: Case communicated to Société de Biologie.

(25) Pitres, A.: "Sur les Atrophies partielles des circonvolutions cérébrales, consécutivement aux amputations anciennes des membres du côté opposé."—'Gazette médicale de Paris,' 1877.

(26) Varigny H. de: "Note sur un Cerveau d'amputé au point de vue de localisations cérébrales."—'Bulletin de la Société anatomique de Paris,' 1879.

(27) Bourdon: "Observations d'un cas d'atrophie du cerveau consécutive à l'amputation d'un membre."—'Bulletin de l'Académie de Médecine,' No. 20, p. 256.

(28) Gowers: "The Brain in congenital Absence of one Hand."—'Brain,' No. III. 1878. p. 388.

CASES OF INJURY TO THE CERVICAL REGION OF THE SPINAL CORD.

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The following cases of injury to the spinal cord have been treated in the Manchester Royal Infirmary since January, 1885, and appear to be of sufficient interest to warrant their collective publication. For permission to make use of them I am indebted to the surgeons under whose care they have been admitted. I have also to thank the house-surgeons and dressers of the Hospital for much assistance in taking the notes, but I am especially indebted to the Resident Surgical Officer, Mr. Joseph Collier, who has had exceptional opportunities of observing most of these patients throughout their stay in the wards, and who has most generously placed his private notes at my disposal. Except where otherwise stated, the post-mortem examinations have been made by Dr. Harris, pathologist to the Infirmary. I may add that the cases are in no way selected, all injuries of the cervical region of the cord which we have admitted during the last eighteen months being here included. We have, of course, had cases presenting slight bruises of the back, it may be with somewhat prolonged pain and weakness, but in none of these were there any symptoms of injury to the cord, and they are therefore not included in the present series. We have had no case of "concussion of the spine" in which spinal cord symptoms supervened on a remote injury, a general shake, or a slight local injury, nor have I ever seen such a case in the Manchester Infirmary.

The cases arrange themselves into two great classes, the one where the diagnosis was confirmed and the symptoms explained by the death of the patient and a post-mortem examination;

the other where there was fortunately no such demonstration of the pathological condition. It will be convenient to consider first the cases in the former group, and then to endeavour, by the light thus gained, to realise more fully the significance of the remainder.

CASE I.—*Fracture-dislocation between the Fifth and Sixth Cervical Vertebræ.—Complete Paralysis of all Nerves below the Fifth Cervical.—Death.*

L. F. was admitted to the Manchester Royal Infirmary under the care of Mr. Jones on June 5th, 1886. The patient was a man, aged 68, a labourer by occupation. On June 1st, four days before admission, he was standing on a short ladder whitewashing, when, two of the rungs of the ladder giving way, he fell backwards in such a position that while his feet remained caught in the ladder, he first struck the back of his neck against a bench some two-and-a-half feet above the ground, and his head then fell to the ground. At the same time he thinks that his bucket of whitewash fell upon him.

He at once became unconscious, remaining so for several hours. On regaining consciousness he found that his legs were quite immovable and his arms partially so, while there was numbness in the lower portion of his body, and insensitiveness to external impressions, coupled with a dull aching pain. There was also great pain in the head, neck, and shoulders. The urine had to be drawn off twice daily. He remained in this condition until his admission to the Infirmary on the fourth day after the accident.

When admitted he was found to present complete paralysis of the lower limbs, and of the abdominal and thoracic muscles. All the muscles of the arms were paralysed, with the exception of the biceps, brachialis anticus, supinator longus and deltoid, the consequence being that the elbows were flexed, the shoulder abducted and rotated outwards, and the hands and arms fell into the position indicated in the annexed engraving (Fig. 1, p. 512), taken from a photograph for which I am indebted to Mr. Sidebottom. There was no power of extension of the forearm, but a fair degree of power of flexion. Some rotation of the humerus could be effected, apparently by the fibres of the deltoid. The contractility of the supra- and infra-spinati was not ascertained; the pectoralis major and latissimus dorsi were paralysed. The lower limbs and trunk were completely anæsthetic as high as the second rib in front,—*i.e.* as high as the descending branches of the cervical plexus—and apparently to the third dorsal vertebra behind. Above this point sensation was normal. The upper extremities were anæsthetic, with the exception of the outer side of thefore arm and hand and the balls of the thumbs. Both cutaneous reflexes and tendon reactions were absent throughout. Breathing was entirely diaphragmatic. There was retention of urine, and the bowels had not been moved since the accident. The penis was subject to frequent erections with seminal emissions; on the back were several bed-sores. The pulse was slow and full; the temperature 98°. The pupils were both somewhat contracted. Over the 5th and 6th cervical vertebræ was a slight prominence, which was very tender on pressure. The only treatment adopted was complete rest on a water bed, catheterisation at intervals, and some simple dressing for the bed-sores.

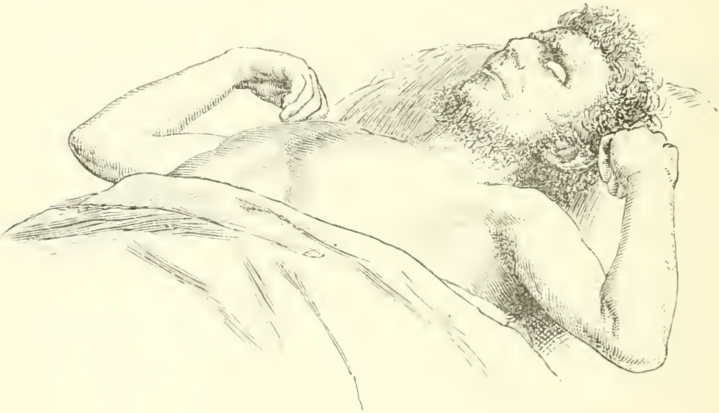

Fig. 1.—Position occupied by the limbs in a case of complete transverse destruction of the spinal cord immediately below the level of origin of the fifth cervical nerves.

During the progress of the case there were not many very important deviations from the symptoms above described. The urine, which was at first healthy, became turbid and alkaline a few days after admission. After this the bladder was washed out daily with boracic lotion. Priapism became more constant. The bowels were never moved except after the use of an enema, or of croton oil. On June 9th, four days after admission, the arms had become completely anæsthetic and remained so throughout. The temperature was generally slightly below the normal; the pulse was often 50, never above 60. Respiration was rapid throughout, being usually about 20, sometimes 30 per minute.

The patient's strength failed steadily, and at times there was confusion of thought, with low muttering delirium at night. He complained mainly of pain in the neck, and of cold feet.

On June 15th he had lost all power of rotating the humerus, on both sides, and passive movement of the shoulder-joints was very painful. The cheeks fell in and out very markedly on respiration. Although the power of the voice remained good, speech was almost unintelligible; he could not protrude the tongue beyond the teeth, nor freely open the mouth. There was marked fibrillar trembling of the masseters; and much accumulation of tenacious saliva about the mouth. There were bed-sores over the sacrum, on the inner side of the right thigh, and on the inner side of the left knee, these having hardly changed since admission.

On June 17th the contraction of the deltoid muscles, which had maintained the abducted position of the arms, ceased, and the elbows fell to the sides, the elbow-joints themselves remaining flexed.

From this date the patient gradually sank, respiration becoming more laboured, and the countenance dusky, while there was increasing apathy. On June 25th the pupils were noted as being dilated and unequal, the right being the larger; there was a commencing "bed-sore" on the chest; on washing out the bladder there came away shreds of what was thought to be mucous membrane. On June 26th he died exhausted.

At the *post-mortem* examination was found depression of the 6th cervical spine and laminæ; the disc between the 5th and 6th vertebræ was broken across, the upper part of the body of the 6th vertebra being tilted backwards. The laminæ were uninjured, but the right superior articular process of the 5th vertebra, and the corresponding transverse process of the 6th were broken off. At this point the cord was compressed, being flattened for a distance of about ¼ inch. The dura mater was here yellowish and opaque, the arachnoid and pia mater healthy. The cord was much softened for a distance of two or three inches above and one inch below the seat of injury, especially around the central canal, where it was quite diffluent and of a yellowish colour, showing under the microscope numerous granule cells. The rest of the cord was healthy. The bladder contained a quantity of blood-stained turbid fluid, its mucous membrane hanging in shreds, beneath which the wall was dark and livid; inflammation had extended to the pelvic cellular tissue and peritoneum. The kidneys were congested, and presented numerous scattered points of suppuration; there was no dilatation of or suppuration in their pelves. The other organs presented nothing of interest.

This case is a sufficiently typical one of injury to the spinal cord above the origin of the 6th cervical nerves. The most interesting feature presented by it is the distribution of the paralysis and anæsthesia. Thus we find that, with the exception of the deltoid, biceps, brachialis anticus and supinators, there was absolute paralysis of the muscles of the upper extremity. The case is thus an exact counterpart of the so-called Erb's paralysis, in which we meet with paralysis of the above named muscles only, and enables us to confirm previous researches as to the distribution of the 5th cervical nerve-root. Besides the intrinsic muscles of the arm there was certainly paralysis of the pectoralis major, which is supplied by the external and internal anterior thoracic nerves, and presumably also of the pectoralis minor. The latissimus dorsi appeared to be paralysed, and no doubt it had lost its nerve supply, from the long subscapular, as well as from the numerous smaller dorsal and lumbar branches, far below the seat of the lesion. That the subscapularis was paralysed appeared to be indicated by the external rotation of the humerus, in marked contrast to the internal rotation seen in Erb's paralysis and in obstetrical paralysis, where this muscle is spared. It was not found practicable to ascertain the condition of the teres major and minor, but the fact that the former is supplied by the middle subscapular would render it probable that it was paralysed with the subscapularis and latissimus, whereas the nerve to the teres minor is the circumflex, and we would therefore expect it to escape with the deltoid. The teres minor would also aid the posterior fibres of the deltoid in producing the well-marked external rotation of the humerus. The serratus magnus, supra- and infra-spinati remain for consideration; but on the innervation of these three muscles we have here no information.

In Erb's paralysis these muscles escape, and would therefore not appear to be supplied by the 5th root; in the closely-allied form of peripheral lesion described by Duchenne as "obstetrical paralysis," the spinati muscles are affected, and it would hence appear that the 4th cervical root supplies them with nerve fibres. Certain it is that they must be supplied by either the 4th, 5th, or 6th cervical, inasmuch as their nerve, the suprascapular, is derived from the cord formed by these three, before its junction with any other root, and as the 6th root is found to supply the triceps, which is not affected in either Erb's paralysis, or obstetrical paralysis, we must refer the innervation of these muscles to the 4th. Thus, then, we arrive at the result, that the 5th cervical nerve supplies only the deltoid, biceps, brachialis anticus and supinator longus (with probably the supinator brevis and teres minor also).

As regards sensation, we find that a cord lesion which cuts off the whole of the nerves of the brachial plexus below the 5th cervical causes anæsthesia of the whole of the upper limb, except the outer side of the arm and forearm, and the radial border of the thumb, *i.e.*, a part of the region supplied by branches of the musculo-spiral nerve. Possibly the area in which sensation was retained was at first more extensive than the above, inasmuch as the case was only seen for the first time four days after the injury, and we find that by the 8th day all sensation was lost in the upper extremity: a change due, no doubt, to extension of myelitis from the seat of injury. To this same zone of myelitis we must ascribe the contracted condition of the muscles supplied by the 5th cervical root, a contraction which slowly gave way to paralysis, as irritation was followed by destruction of the grey matter of the cord.

The affection of speech and other symptoms noted in the progress of the above case, at first gave rise to the supposition, that there might be some extension of myelitis to the medulla oblongata; but as no further symptoms of bulbar paralysis developed themselves, and as the intermediate cervical muscles were unaffected, these phenomena would appear to be due to the general debility only.

CASE II.—*Fracture-dislocation between the Fourth and Fifth Cervical Vertebræ.—Complete Paralysis of Limbs and Trunk.—Death.*

The following case was under the care of Mr. Heath. The notes were taken by the dresser, Mr. W. H. Iddon.

M. L., male, aged 45, was admitted on March 27th, 1886. He had fallen from a scaffolding some forty feet in height. In addition to three lacerated scalp-wounds, he presented the following symptoms. There was severe pain in the back of the neck. All four extremities were completely paralysed, as were the muscles of the trunk and abdomen. The limbs and trunk were absolutely anæsthetic below a line running across the thorax at the level of the second intercostal space, and thence across the deltoids at the junction of their upper and middle thirds; above this line sensation was normal, the transition being quite abrupt. The pupils were equal, and appeared to be of normal size. There was no mental affection. Respiration was diaphragmatic; the urine was retained; priapism was constant. Death took place a few hours after admission, apparently from dyspnœa.

The temperature was taken several times, but was unfortunately not recorded. The house surgeon assures me that it was certainly not, at any time, far from the normal.

At the *post-mortem* examination there was found a fracture through the body of the 5th cervical vertebra, and the intervertebral disc immediately above it, The spines of the 3rd, 4th and 5th vertebræ were broken off. Outside the dura mater at this point was a very slight effusion of blood. The cord was much lacerated, being almost torn across opposite the seat of fracture, and was very soft for a distance of nearly 1½ in., being reddened by extravasated blood. The skull and its contents were uninjured. The lungs were œdematous.

The above case contrasts well with Case I. Here we have the lesion affecting the 5th vertebra and the disc immediately above it, so that the 5th root, which escapes from the spinal canal above the 5th cervical vertebra, would be injured, as well as all parts below. Hence we get complete paralysis of sensation and motion in the upper extremity, the whole of the brachial plexus being cut off from the brain, with the exception of the branch of the 4th cervical nerve. The present cases unfortunately yield no evidence as to the distribution of this branch, the condition of the posterior scapular muscles not having been specially noted.

Case III.—*Dislocation between the Fourth and Fifth Cervical Vertebræ. —Complete Paralysis of Limbs and Trunk.—Death.*

C. D. was admitted under the care of Mr. Hardie. The notes were taken by Mr. Benson, House-surgeon. The patient was a carter, 39 years of age. He was admitted on August 3rd, 1886, having fallen from a van, of which the wheels had passed over his shoulders.

On admission he complained of great pain at the back of the neck. All four limbs were completely paralysed, and there was anæsthesia below the level of the descending branches of the cervical plexus. Respiration was diaphragmatic. The fæces were passed unconsciously. Both pupils were contracted, the palpebral fissures narrowed, and the eyeballs presented a remarkable sensation of softness owing to paralysis of the posterior orbital muscles. Pulse was full, of low tension, and beating at the rate of 60 per minute. The temperature on admission, which was in the evening, was 96·8°.

He passed a restless night, and on the next morning the following additional symptoms were noted. There was constant priapism. The urine was retained, and great difficulty was found in passing a catheter, due apparently to the extreme dryness of the urethra, as it entered readily after an injection of oil. The urine was examined for sugar and albumin, with negative results. The face was much congested; the pulse remained full and soft, beating at the rate of 130 per minute. The temperature at 8 A.M. was 102·2°. An interesting fact noted by Mr. Collier was, that when the patient was told to take a deep breath, he produced contraction of the levator anguli scapulæ muscle, which could be distinctly felt beneath the trapezius, thus indicating an attempt to fix the scapula, and bring into play the extraordinary muscles of inspiration.

At 10 A.M. the patient's temperature had risen to 103·6°; at midday to 105°, at 2 P.M. to 106°. At 4 P.M. it was 107·8°. He now became insensible and livid, and the breathing more shallow. The whole body was covered with a profuse hot sweat. At 5 P.M. he died, the temperature having risen to 108°. The temperature after death was not recorded.

At the *post-mortem* examination there was found a rupture of the cartilage between the bodies of the 4th and 5th cervical vertebræ, these two bodies being so widely separated behind that a finger could be introduced between them, They thus formed an obtuse angle posteriorly, pressing on the contents of the spinal canal. At this point there was marked post-mortem staining of the meninges, but decomposition was so far advanced that it could not be ascertained whether they were inflamed. The cord itself was here compressed, through a vertical extent of $\frac{3}{4}$ of an inch, into a narrow band, and was very much softened, there being no distinction between the grey and the white matter. Elsewhere the cord showed only post-mortem softening.

The posterior parts of both lungs were much congested, and in the brain were numerous puncta cruenta. There were no other changes of interest.

This case is similar to the last, the lesion again involving the 5th cervical nerve and all parts below it, and the symptoms being the same. We shall refer again to such further points of interest as it presents.

CASE IV.—*Fracture-dislocation between the Fourth and Fifth Cervical Vertebræ.—Partial Paralysis of Right Upper Limb.—Total Paralysis of other Limbs and Trunk.—Death.*

T. L. was admitted under the care of Mr. Heath on Sept. 7th, 1886. He was 36 years of age, and a carter by occupation. While attending to his horse the animal had fallen against him, crushing him against a manger, the corner of which struck his back between the shoulders. He immediately fell down paralysed.

On admission there was found no external bruise or other evidence of injury, except a sense of soreness and stiffness in the lower cervical spine; no irregularity of the spinous processes could be detected. The lower limbs and trunk muscles were entirely paralysed, as was the left upper extremity; the right upper extremity was paralysed with the exception of the deltoid and biceps (the condition of the brachialis anticus and supinator longus is not noted). The bladder and sphincter ani were also paralysed, and there was priapism.

There was anæsthesia of the lower limbs and trunk, and of the upper limbs with the exception of the back of the hand, forearm and arm on both sides. Breathing was entirely diaphragmatic. The pupils were widely dilated. The temperature was 100·6°. The treatment consisted in the use of a water bed with ice-tubes to the spine, doses of ext. ergot. liq., three times daily, and catheterisation.

At 8 A.M. on the following day the temperature had risen to 105·2°; at noon it was 104°; and then again gradually rose, reaching 105·6° before death, which occurred on the second day. Breathing became more difficult, with accumulation of mucus in the bronchial tubes, and the face grew very livid. He died asphyxiated about thirty-six hours after admission, the heart continuing to beat for some ten minutes after respiration had stopped. An unsuccessful attempt was made to save the patient by bleeding from the arm, artificial respiration, and stimulation with the faradic current.

At the *post-mortem* examination there was found a rupture of the cartilage between the 4th and 5th cervical vertebræ, "which fracture had extended across posteriorly, involving the laminæ of the 4th or 5th vertebræ." The spinal membranes were healthy. The cord opposite the seat of fracture was not at all compressed, but was for a distance of about an inch very soft and pulpy, containing numerous punctiform hæmorrhages, which were most marked in the central grey matter. The other organs were not examined.

This case presents an interesting comparison with Case I. on the one hand, and with Cases II. and III. on the other. The lesion of the spine itself was less severe, and there was less displacement of the bones, which consequently did not cause permanent compression of the cord. There was, however, ample evidence of a temporary crush of the spine, received, no doubt, at the time of injury, the bones afterwards recoiling partially to their normal position. Such, probably, is the explanation of many cases (as, for instance, our sixth case), where there is little or no evidence of bone lesion, but the cord itself has sustained a severe injury. An accident causes an acute bend of the vertebral column, which at once rights itself, but not before great or irreparable damage has been inflicted upon the contained organ; and many of these cases have been regarded as instances of "concussion of the spinal cord," the possibility of a gross mechanical lesion being overlooked. Another point which is here exemplified, but which is more fully demonstrated in Case VI., is the tendency for hæmorrhage into the spinal cord to affect mainly its central portions, where its substance is softest and where the large branches of the central spinal artery have their distribution.

As regards the distribution of the paralysis and anæsthesia in this case, we find that there was not complete annihilation of the functions of the two 5th cervical nerves. On the left side, the upper limb was completely paralysed, as in Cases II. and III., but on the right side the muscles supplied by the upper root of the brachial plexus had partially escaped, the biceps and deltoid not being paralysed. As regards the supinators and the brachialis anticus we have no information, and I did not see the case, but should suppose that these also probably escaped, although it is quite conceivable that their spinal motor cells may lie a little lower in the cord and may thus have been involved in the lesion. Further, we find that as in Case I., so here, sensation was retained in the distribution of branches of the musculo-spiral nerve, on the outer border of the arm, forearm, and hand of both sides.

CASE V.—*Comminuted Fracture of Fifth and Sixth Cervical Vertebræ.—Partial Paralysis of Left Upper Limb.—Total Paralysis of other Limbs and Trunk.—Death.*

W. H. was admitted to Mr. Whitehead's wards on April 30th, 1886. He was 28 years of age, a wine-merchant by occupation, and of intemperate habits. When admitted he was intoxicated, and had, while in that condition, fallen over the bannisters of a staircase, the distance not being ascertained. He complained of great pain at the back of the neck; but there were no external signs of injury. There was complete paralysis of both lower extremities and of the right upper limb; but on the left side he could bend the elbow-joint. Respiration was diaphragmatic. The cutaneous reflexes and tendon reactions were abolished. Anæsthesia was complete below the branches of the cervical plexus, except over a portion of the left upper limb. The pupils were moderately contracted, the contraction—or rather imperfect dilatation—showing best, as is usual in these cases, in diffused light: the palpebral fissures were small. The pulse was slow and compressible. The temperature was not recorded, but was below the normal. The urine was retained, and the penis large and turgid.

A more complete examination showed, that while there was absolute loss of motion and sensation in the right upper extremity, the paralysis had on the left side spared the deltoid, biceps, brachialis anticus and supinator longus; the pectoralis major and latissimus dorsi, and all the muscles below the shoulder, with the exception of those mentioned, were paralysed. The arm occupied exactly the position assumed by those of Case I., being slightly abducted at the shoulder, rotated outwards, with the elbow flexed and the forearm and hand supine, the contrast between this position and the complete flaccidity of the right upper extremity being very striking. This was the first case in which this remarkable position of the limb was noted in the Manchester Infirmary, the detection of its significance being due to Mr. Collier. Its importance is shown by the fact, that it was found possible to ascertain the exact nature of the cord lesion before death. Anæsthesia extended over the whole of the left upper extremity, except a strip of skin, some three inches wide, extending downwards from the shoulder, along the outer side of the limb to a point about three inches below the level of the elbow-joint.

The patient complained much of difficulty of breathing, which increased rapidly, and he died asphyxiated about forty hours after the accident. Before death the temperature became very high.

At the *post-mortem* examination, which was made by Dr. Bury, Medical Registrar, there was found a complete crush of the body of the 5th cervical vertebra, the 4th being displaced downwards and forwards, with its left transverse and articular process broken, and the 6th split vertically: the condition of the bones is shown in Fig. 2. In front of the bodies of the injured vertebræ was some extravasation of blood. On opening the spinal canal, the cord was found to be obliquely compressed in such a way that the upper limit of the flattening was above the 5th cervical nerve on the right side, but between that and the 6th nerve in the left, as shown in Fig. 3 (p. 522), a small piece of the body of the fifth vertebra pressing upon the 5th root of the right side. The lungs were intensely congested, and portions of the left lower lobe sank when thrown into water. The other organs presented nothing of interest.

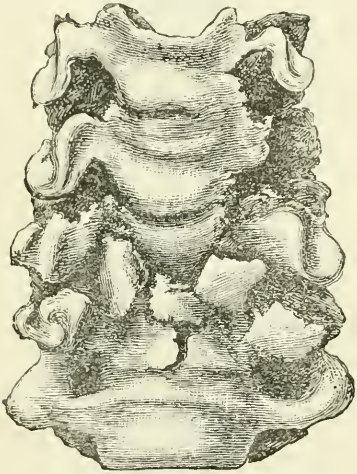

FIG. 2.—Third, fourth, fifth, sixth and seventh cervical vertebræ from Case V., seen from the front, showing a comminuted fracture of the bodies of the fifth and sixth vertebræ.

This case, like the preceding one, is an instance of oblique injury to the cord, the direction of the obliquity being reversed, and the 5th root escaping on the left side only; and it thus offers another illustration of the distribution of the motor and sensory branches of this root. The area in which sensation was retained was less than in Cases I. and IV., owing probably to some of the lower fibres of the left 5th root also being involved in the lesion.

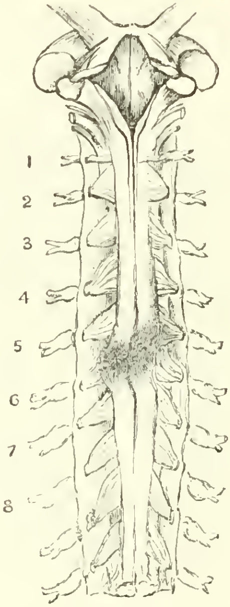

Fig. 3.—Spinal cord from Case V., seen from behind, showing compression, extending obliquely from above the origin of the sixth cervical nerve on the left side, upwards, so as to intercept the fifth nerve on the right side.

The above have been cases of fracture of the cervical spine, but the next on our list differs in that the lesion was not a fracture, but a traumatic hæmatomyelia, there being no obvious lesion of the spinal column itself.

Case VI.—*Traumatic Hæmatomyelia in Lower Cervical and Upper Dorsal Region.—Death.*

J. B., aged 34 years, was admitted on December 30th, 1885, under the care of Mr. Heath, suffering from an injury to the spine. He was a carter by occupation, and was hurt, while loading a waggon, by a "tippler" full of coal falling upon him, so as to throw him upon his face, the coal striking him between the shoulders.

On admission to the Infirmary he was quite conscious, but stated that he had been rendered insensible for some time by the injury. He had complete loss of power over both lower extremities, and complained of great pain at the back of the neck and between the shoulders, which was increased by moving the head. He had also pain from a severe bruise over the right elbow. On the left side of the head were three slight scalp-wounds.

A more careful examination showed him to have pain mainly over the 5th and 6th cervical vertebræ, increased by pressure; but no irregularity was here detected in the spine. There was absolute paralysis of both lower extremities, with deficient action of the intercostal and anterior abdominal muscles in respiration, but good movement of the diaphragm. Both legs were completely anæsthetic as high as the knees; but thence upwards he had some sensation, although there was distinct numbness as high as a line drawn round the abdomen about two inches below the umbilicus. There was no hyperæsthesia. The plantar reflexes were noted as "almost absent," and the feet felt very cold, with some prickling sensation. He had distinct priapism. Both pupils were much contracted, but equal and contracting further on exposure to light. There was no vomiting and no mental affection. He was placed on a water bed, the scalp-wounds dressed, and ext. ergot. liq. administered in ʒss doses. In the evening the urine had to be drawn off.

On the following day there was still absolute paralysis of the lower limbs; but there was now no anæsthesia. The priapism had subsided, and the urine was passed without any trouble. The diaphragmatic respiration was very marked. On examining the arms, which had not been done the day before, I found distinct feebleness of the flexors of the wrist and fingers on both sides; the grip was very feeble, but the power of extension against resistance little if at all diminished. Both superficial reflexes and tendon reactions were everywhere absent. The left pupil was somewhat contracted, but the right one very much more so; both contracted further on exposure to light. The patient had some vomiting during the night, and there were râles all over the chest. The morning temperature was 98·6° F.; in the evening it was 99·8° F.

On the following day, January 1st, 1886, his fæces were passed involuntarily. There was severe and painful cough, and all over the chest loud crackling râles; the countenance was cyanotic.

No other change had occurred. Temp., morning, 99°; evening, 100° The cyanosis and difficulty of breathing now rapidly increased, and there was dulness at the bases of both lungs, but no crepitations were heard. Without further symptoms, the man died on the night of January 2nd, 1886.

At the *post-mortem* examination there was found engorgement of the bases of both lungs and of the right side of the heart; but the other organs were healthy, with the exception of the spinal region, which is thus described. "A small dark black hæmorrhagic effusion existed in the muscles over the lower cervical and upper dorsal regions." The membranes of the cord were quite normal, and the external appearance of the cord itself presented no evidence of injury; but "on section there was found to be a dark black hæmorrhage into the central grey matter, in the lower cervical and upper dorsal regions. This hæmorrhage, which measured in its vertical extent from 1½ to 2 ins., was in the greater part of its extent situated centrally, occupying the whole of the central grey matter and extending but little into the white substance, which in its neighbourhood was merely softened and of a faintly yellow tinge. At the lower part, for a very short distance, the hæmorrhage was limited to the anterior cornu of the right side, while the corresponding left horn appeared to be perfectly healthy. Elsewhere the cord was firm, and presented no abnormality."

The above case presents a large number of points of interest. In the first place, as regards the pathology, we see here, as in Case IV., the tendency for hæmorrhages in the cord to affect the region immediately surrounding the central canal, where the vascular supply is very free. But the question which at once presents itself is, how are we to explain the occurrence of this hæmorrhage without an injury to the vertebral column? It appears to us impossible to suppose that it was due solely to concussion—to an indirectly-transmitted shake or jerk of the cord. If such were the method of its production, and if it were due to the cord being jerked forwards against the bodies of the vertebræ, we should expect the post-mortem evidence of the bruise, thus given to that delicate structure, to extend over a wider area: the cord would, in such an injury, be thrown forwards without any acute bend, and we should expect to find, not a single localised hæmorrhage, but a more diffused and extensive lesion. It appears far more probable that the mode of production of the injury was a sharp bend of the vertebral column at the region where it was struck—the blow being, as we have seen, a direct and severe one—the bones at once recoiling, but the more delicate cord being partially ruptured at the point of this sharp bend. A condition somewhat similar to this is seen in Case IV., where, however, the spinal column shows the effects of the strain to which it has been submitted, although it has recoiled sufficiently to obviate permanent compression of the cord. In the present instance we have only to suppose the injury to the framework to have been a little less severe than in Case IV., and the crush to its contained viscus to have been practically the same. This explanation of a temporary bend of the spine, with consequent injury to the cord, is advanced by Mr. Page in the July number of 'BRAIN,' as the true pathology of MM. Duménil and Petit's case of "concussion" there described; and we would suggest that the present case not only confirms Mr. Page's view, but that the case which he quotes was probably of exactly the same nature as the one at present under consideration. Had our case survived for thirty-five days, as did that of MM. Duménil and Petit, we should have expected to find exactly the appearances described by them, viz. a patch of softening and discoloration at the point struck, with probably a secondary degeneration of the lateral columns below that region.

If, however, we are to assume the present case to be one of direct injury and not of "concussion" of the cord, it will be necessary for us to explain the various symptoms produced by this comparatively slight lesion, and this there appears to be no difficulty in doing. It is well recognised that in the case of cerebral hæmorrhage, we meet with two classes of symptoms, viz. those due to the actual destruction of a portion of the brain substance by the effused blood, which are permanent; and those due to compression of surrounding tracts or nuclei, which are more or less evanescent, their disappearance being conditional on the absorption of the blood and consequent diminution of the compressing force. Thus it may be and often is the case, that the "compression symptoms" entirely overshadow the "destruction symptoms," and that a comparatively trifling hæmorrhage may cause extensive and more or less persistent compression of important portions of the brain. Precisely similar appears to have been the condition in the present case. The hæmorrhage itself occupied only the region of the central canal in the lower cervical and upper dorsal portions of the cord, extending to but a slight extent into the right anterior cornu. But this effusion of blood would suffice to cause more or less pressure upon the whole of the cord at this level, a pressure to which probably that structure would soon accommodate itself, having, as it has, ample room to expand. Perhaps then it will not be too bold an assumption to suppose that the direct symptoms, due to destruction of the grey matter by the blood, were insignificant; the other and more serious results of the injury arising from mere compression by this effusion.

There is no difficulty in attributing the paralysis of the lower part of the body to compression of the descending columns of the cord. The anæsthesia was partial only, becoming somewhat gradually less marked towards its upper limit, and this would coincide with the view, that the sensory fibres for the lower part of the body are more centrally situated in the spinal cord: a condition obviously convenient in view of the fact, that they have a longer course to travel. If this be their arrangement, we should expect exactly the condition which we find, viz. a more severe compression, and consequently more absolute loss of conduction of the deeper fibres, those most superficially situated having apparently escaped altogether. Again, in the case of the arm muscles, we find that the extensor muscles of the wrist and fingers have apparently escaped entirely, whereas the flexors are partially paralysed. Now, the hæmorrhage, extending as it did to the lower cervical region, would compress the anterior cornua in that region, and it is well recognised that it is from this portion of the cord that the flexors of the wrist receive their nerve supply: these muscles are not completely paralysed, as would be the case in a destroying lesion of their nuclei, but partially so, as we should expect in a compression lesion. On the other hand, the extensors of the wrist and fingers derive their nerve supply from a somewhat higher point in the cord, probably from the 6th or 7th cervical nerve, and these muscles are little, if at all, affected, while the muscles, already so often mentioned, which are supplied by the 5th cervical root, certainly escaped entirely. Finally, the early diminution of the anæsthesia, and of other symptoms such as the priapism and retention of urine, would well accord with the theory of their production by a compression of the conducting fibres, to which the latter rapidly accommodate themselves. On these grounds then we hold, that in no sense can the above case be regarded as one of concussion of the cord, but rather as one of a direct blow causing a small local hæmorrhage, with important symptoms arising from the compression thereby induced.

We now leave the region of diagnosis by post-mortem examination, and pass on to the consideration of some cases of recovery after injury to the cervical spine.

CASE VII.—*Traumatic Hæmatomyelia in Middle Cervical Region. —Recovery with Persistent Symptoms.*

W. H., aged 36, a carter, was admitted under the care of Mr. Hardie on June 13th, 1885. About half-an-hour before admission he had fallen between the shafts of a van which he was driving, one wheel of which had passed over his shoulders. It may be remarked that there was no history of syphilis.

On admission he lay quite helpless, there being complete paralysis of the left arm and of both legs, while the right arm was partially paralysed, but retained some motor power "especially at the elbow." Respiration was diaphragmatic. The reflexes were normal. There was anæsthesia below the distribution of the cervical nerves. Speech was laboured, owing to difficulty in breathing, but there was no loss of consciousness or mental confusion. The left pupil and palpebral fissure were slightly smaller than those of the right side. There was much pain in the back of the neck, but no abnormality was there detected. After he had been put to bed, he was able to move the right lower extremity slightly. He was placed on a water bed, the head being fixed by sand-bags. There was great thirst and dryness of the mouth.

The urine had to be drawn off; it was acid, of a specific gravity of 1010, and contained neither sugar nor albumin. He passed a fairly good night.

On the following morning it was ascertained that sensation was nowhere absolutely lost, but that it was impaired on both sides of the body, but more markedly on the right than on the left. There was more power of moving the right leg and arm; the limbs of the left side remained completely paralysed. Other symptoms continued as before; the temperature was, in the morning, 100°·2 in the evening, 101°. There was free perspiration, the urine was scanty and high-coloured.

When examined on June 15th, forty-eight hours after the accident, he was feeling much better and had less pain in the neck. He had more power over the right arm and leg, and an examination of the arm showed that the rhomboids, posterior scapular muscles, deltoid, biceps and supinator longus acted well, whereas the other muscles of this limb were paralysed. The left arm and leg remained completely paralysed. Sensation was returning on both sides, but remained less acute on the right than on the left side. This diminution of sensation extended as high as the supraclavicular branches of the cervical plexus. The reflexes were normal. There appeared to be some slight movement of the intercostal muscles in respiration. The pulse was beating at the rate of 102; the temperature was, in the morning 102°; in the evening, 102°·4.

On the following day there was further gain of sensation and of power on the right side. On the left side were slight pricking sensations. The temperature had fallen to 99°·4. Other symptoms were as before. It was noted that there had throughout been no priapism.

On June 17th he passed his urine without the use of a catheter, and had no further trouble with it. His bowels were now opened for the first time since the accident. The pulse remained somewhat rapid, being 85 in the morning and 100 in the evening; the temperature was 101°. On the 19th there was distinct costal respiratory movement. The pulse was still rapid and the temperature 100°. On the 26th the temperature for the first time reached the normal, at which it afterwards remained, except temporarily on two unexplained occasions.

On June 24th there seemed to be little or no further change. He was ordered iodide of potassium in gr. 10 doses. From this point improvement progressed very slowly. On July 4th he was able to move his shoulders slightly on both sides, and the intercostal muscles acted more freely on the right than on the left side; there was no other change in the paralysis. The knee-jerk was exaggerated on both sides; there was no ankle-clonus. On July 20th he was transferred to the medical wards under the care of Dr. Ross, where very full notes of his condition were taken by Dr. Bury, Medical Registrar, of which an abstract only is here given. At this time it was noted that costal respiration was still deficient, being apparently more so in the lower ribs (probably because the downward traction of the diaphragm was able to overcome the expansion).

The following was the condition of the right upper limb:—The wrist was semiflexed and somewhat inclined towards the ulnar side, having very slight power of flexion and extension. The thumb lay fully extended, but could be slightly flexed, adducted and abducted: there being, however, no power of opposition. The first phalanges of the fingers were extended, the second and third partially flexed; there was slight power of flexion of the metacarpo-phalangeal joints, but no movement at the inter-phalangeal joints. As regards lateral movement there was slight power of abduction and adduction of the first and fourth fingers, but the second and third were immovable, having always a slight interval between them. He could pronate the wrist and then bring it half-way into the supine position, but not beyond this. The elbow was semiflexed and could be partially flexed, but not extended and passive extension caused the tendon of the biceps to stand out very sharply. At the shoulder-joint there was diminished movement, especially of abduction, external rotation and flexion; he could touch the tip of the opposite shoulder but not his head. There was marked wasting of the whole limb. A very powerful faradic current caused slight contraction of the extensors and flexors of the elbow, and of the serratus magnus, but of none of the other muscles of the limb or shoulder. The galvanic current gave the "reaction of degeneration" with the pectoralis major, deltoid, supra- and infra-spinatus, biceps, triceps, extensors and flexors of the forearm and muscles of the hypothenar and thenar eminences. On the left side the condition was similar, except that there was even less power of voluntary movement. In brief, there was well-marked "atrophic paralysis" of all muscles supplied by the brachial plexus. The movements of the lower limbs were weak, but of normal range, except for some deficiency at the left ankle, and the electric reactions were also here normal.

It was not easy to map out fully the extent of sensory deficiency, owing to the fact that there was nowhere absolute anæsthesia, nor any defined limit. On both sides sensation was normal in the neck, and as far downwards as the lower limit supplied by the cervical plexus, that is, to the level of the third rib. There was below this limit marked diminution of sensation on the right side extending to within ½ inch of the middle line in front: at this point there was an area of transition, and to the left of the middle line sensation was normal. Behind, the anæsthetic area extended over the whole of the right side of the trunk as far as a vertical line, midway between the posterior border of the right scapula, and the spines of the vertebræ; the upper limit of anæsthesia being here the spine of the scapula. The right shoulder was not anæsthetic. There was no anæsthesia of the left half of the trunk. The right lower limb was anæsthetic, the left was not—in fact, there was partial anæsthesia of the whole of the right side of the trunk and lower extremity, with normal sensation on the left side. In the upper limbs the distribution of anæsthesia was symmetrical, affecting the whole of the limbs, excepting a strip of skin, running from the shoulder, along the centre of the biceps, and then straight downwards along the front of the forearm to the hand, where it spread out, so that the only part of the hand which was anæsthetic was the thumb, the rest having apparently normal sensation both in front and behind.

The knee-jerk was much exaggerated, and there was marked ankle-clonus; the superficial reflexes were absent, and there were no reflexes in the upper limbs.

The skin over the anæsthetic areas was hot and dry, the slightest prick or scratch causing a bright red spot or line. There was slight swelling and tenderness at the back of each wrist. The inter-phalangeal joints were also thought to be slightly swollen, but were probably merely rendered more prominent by the general atrophy. The finger-nails were long, the skin at their roots being red, smooth, and shining.

The left pupil was slightly smaller than the right, and the left palpebral fissure somewhat narrowed.

The patient remained under observation for rather more than three months after this, taking a mixture of iron and quassia. During this time voluntary power increased considerably in the right upper extremity, and to a slight extent in the left; when last seen he could move the fingers of the right hand fairly well in all directions, had slight power of opposition of the thumb, could raise the elbow to the level of the shoulder, and could touch the top of the head with his hand. Sensation had also improved. There was still some difficulty in moving the left ankle, but otherwise the movements of the lower limbs were good.

Superficial reflexes were obtained over the abdomen; the cremasteric reflexes were still absent, the plantar was got on the right side only. The knee-jerk was less marked on the right side than it had been, but was unaltered on the left. There was no other change, and the vital functions were well performed.

Regarded in the light of the preceding cases, there can be little difficulty in following the course of events in the present instance. The primary lesion was probably a hæmorrhage into the cord situated similarly to that of Case VI., but of somewhat greater extent. On the left side it appears to have extended sufficiently high to annihilate, either by pressure on or by destruction of the grey matter, the whole of the functions of the nuclei connected with the brachial plexus of that side. On the right side the nuclei of the 5th cervical nerve have escaped, as have the pupillary fibres. The parts of the cord below this point have also been affected, probably owing to compression of the conducting tracts, and thus we find partial anæsthesia, which undergoes gradual improvement; and retention of urine, which soon passes off. It is noticeable that this is the only one of our cases of cervical injury in which there was no priapism, and that in Case VI., which was also an instance of mere temporary compression of the conducting paths, priapism was of short duration. In the present instance, as in Case VI., there is paralysis of the lower part of the body, owing to pressure on the lateral columns, and at a later stage we have clearly developed the symptoms of descending degeneration of those columns. The simplicity of the case is, however, interfered with apparently by a localised myelitis arising round the hæmorrhage and thus causing some extension of its destructive effects. Hence in the later stages of the case we find the 5th cervical nerve losing its functions on the right as well as on the left side. That the left side was the more severely injured is evidenced by the fact, that the anæsthesia was more complete and more permanent on the right side of the body. It will also not escape notice that, whereas the muscles supplied by the brachial plexus presented the "reaction of degeneration," those of the rest of the body did not, thus evidencing farther a "destroying lesion" of the grey matter of the cord in the lower cervical region on the one hand, and a mere interference with conduction to the parts below on the other. In the same way we find that in the case of the brachial plexus, sensation is impaired on both sides, whereas, below this point, the anæsthesia is limited to the right half of the body, corresponding with the obviously greater extent of the lesion on the left side of the cord.

The most difficult point in this case is to explain the localisation of the anæsthesia in the upper extremities. This loss of sensation affects both limbs symmetrically, involving two areas, viz. the outer side of the limb, supplied by the 5th cervical root, and the inner side, supplied by the 8th cervical and 1st dorsal, but omitting the intermediate portions which derive their sensory nerve supply from the 6th and 7th cervical roots. The explanation of this remarkable localisation, given by Dr. Ross, under whose care the patient was placed, and for whose inspiration upon both this and other subjects I cannot sufficiently express my indebtedness, is as follows: That the hæmorrhage did not extend far, if at all, below the level of the 5th root, the atrophic paralysis below this point being due rather to subsequent myelitis, which would produce less serious mischief, and would affect mainly the grey matter of the anterior horns. That thus we have, at or about the level of the 5th cervical root, a destroying lesion affecting nearly all the structures of the cord, but that below this point the conducting fibres largely escape. That the sensory fibres of the 5th nerve are injured immediately on their entry into the cord. That the fibres of the 6th and 7th roots, crossing obliquely as they do, and probably very superficially, are, at the level of the 5th roots, so near the periphery as to escape injury; but that the fibres of the 8th cervical and 1st dorsal nerves have probably already completely crossed over before reaching the level of the 5th root, and having thus acquired a deeper site have again come within the influence of the lesion. The great difficulty which this ingenious theory presents is that, on the left side of the body, there is no anæsthesia below the level of the 1st dorsal nerve-root, and that thus we have not sufficient evidence of compression or other injury of the sensory conducting paths on the right side of the cord. Possibly the explanation may be that the hæmorrhage or subsequent myelitis had taken a somewhat irregular direction, extending into the nuclei of some of the roots but not of others; or that, as in Case IV., there were two or more scattered hæmorrhagic foci.

We have only to note further, that here, as in Case VI., the cause of the intra-medullary hæmorrhage was apparently an acute bend of the cord, due to the direct application of great violence.

CASE VIII.—*Traumatic Cervical Hæmatomyelia.—Temporary Paralysis of Limbs and Trunk.—Recovery.*

W. B., aged 40, was admitted to Mr. Heath's wards on Aug. 15th, 1885, suffering from the results of a fall estimated at a distance of 50 feet. He was a strong, healthy-looking man with no trace of previous illness. On the right side of the head was a deep scalp-wound, extending about 4 inches backwards from the forehead, and surrounded by much bruising. It may at once be said that under ordinary treatment this wound soon healed up. There was no unconsciousness, nor other trace of cerebral symptoms. All four limbs were, however, paralysed; the arms completely so; the legs could be moved in bed, but the patient could not stand. He had complete anæsthesia of the arms, abdomen and legs, but no loss of control over the rectum and bladder. The skin over the lumbar spine showed extensive bruising. Unfortunately, the notes of this case were very imperfectly taken, and there is no further information contained in them.

On the following day the temperature rose to 99·2° F., in the morning, and 100·8° F., in the evening: there was more power in the lower limbs and muscles of the neck. On Aug. 17th, he could move his legs quite easily and freely in bed, and there was no sign of paralysis of the neck, but both arms remained completely paralysed. Sensibility was improved, but there were tingling sensations in all the limbs. The temperature was lower. On the 18th sensibility appeared to have quite returned. The fingers and thumb could be moved fairly well on both sides, and there was slight movement of the whole upper limb. The temperature had returned to the normal, at which it afterwards remained. From this time there was a slow but steady gain in power, and on Aug. 28th, the upper extremities could be moved in any direction, but only feebly so. It was, however, some time before the legs were sufficiently strong to support him. He was first able to walk a little on Oct. 12th, and could then use his arms for such light work as holding a book, but they were still weak; there was no marked muscular wasting—merely the emaciation due to lying in bed; there was marked ankle-clonus, and exaggerated patellar, triceps and radial reflexes. The only affection of sensibility was some numbness of the inner sides of the calves, with tingling sensation in the fingers.

Three days later he was sent to the Convalescent Hospital at

Cheadle, where the constant current was applied to various parts of the limbs three times weekly, and where strength rapidly returned.

I again saw the patient on Jan. 16th, 1886, *i.e.* five months after his accident. He then looked very well and had returned to his work, but still felt weakly and had the sensation of tingling in the fingers; there was, however, no anæsthesia, and no difficulty in using the hand. He stated that at times he had some girdling sensation, which he referred to the region of the lower dorsal and upper lumbar nerves. The patellar and radial "deep reflexes" were exaggerated, but there was no ankle-clonus and no jerk on striking the triceps tendon.

The symptoms of this case are entirely explicable on the supposition, that the patient had a hæmorrhage into the cord, affecting the central grey matter at the level of the brachial plexus. This appears to have been small, and to have rendered itself obvious only by causing compression. There was no priapism, and no affection of the bladder or rectum, so that conduction to and from the lumbar centres was even less interfered with than in the two preceding cases. Anæsthesia also was very temporary, thus showing once more how rapidly the sensory fibres of the cord may accommodate themselves to slight pressure. It is interesting to note that as the anæsthesia passed off there was developed some "tingling" sensation, which we saw also in the last case under analogous circumstances. Here also, as in Case VII., we have some descending degeneration of the pyramidal tracts, due probably to pressure, and evidenced by the spastic symptoms in the lower limbs. That the effusion of blood was small, and did not extend to the anterior cornua, is indicated by the fact that there was no atrophy in the muscles of the upper limb. Unfortunately, the circumstances of the accident do not, in this case, allow of our exactly determining the nature of the injury to the spine, but it seems at least probable, from the presence of the severe scalp-wound, that the man fell upon his head, and that then there was an acute bend of the spine in its most vulnerable site, the whole weight of the body acting as a lever to bend the spinal column at this point. The bruise over the lumbar spine was clearly due to a less severe blow than that which caused the scalp-wound, and probably resulted from the body striking the ground after the head. This is precisely the explanation given by Mr. Page of MM. Duménil and Petit's case of "concussion" already quoted; and, indeed, the last three of our cases appear to me to confirm most strongly the views set forth by Mr. Page in the article above referred to.

Case IX.—*Traumatic Meningeal Hæmorrhage (?).—Paralysis of Limbs and Trunk.—Partial Recovery.*

M. H., a widow, aged 37, was admitted under the care of Mr. Jones, on May 10, 1886. Five weeks previously, she received two bullet-wounds from a revolver, probably fired at a distance of about two yards. One bullet pierced the left thumb and then passed through the lobe of the left ear, grazing the mastoid process and apparently at once escaping. The other entered immediately below and behind the left mastoid process, passing downwards and to the right. She was "immediately" paralysed in all four limbs, and when seen by a medical man was found to have complete loss of sensation below the neck, diaphragmatic breathing, and incontinence of urine and fæces. An attempt was made to find the bullet, but proved unsuccessful. The wounds healed well in about a fortnight, during which time the nervous symptoms were said to have remained unchanged. Calomel was occasionally administered to regulate her bowels, and her limbs were rubbed with oil.

On admission to the Infirmary five weeks after the injury, she was found to be a well-nourished and healthy-looking woman. In addition to the scars left by the above-mentioned wounds, she presented the following symptoms. At the back of the neck, some 3 inches to the right of the 5th cervical spinous process, was felt a hard nodule, apparently the bullet, lying quite superficially. She felt pain on moving the head, and tenderness of the cervical spine. There were no cerebral symptoms. The upper limbs were completely paralysed. The lower extremities were less absolutely so: on the left side she had some power of extension and flexion of the ankle-joint, and could raise the limb slightly from the bed; on the right side there was even less movement of the foot. The abdominal and intercostal muscles were also paralysed, breathing being diaphragmatic. There was no anæsthesia, but slight hyperæsthesia about the shoulders. The superficial and deep reflexes were normal. The limbs did not appear wasted. There was incontinence of urine and fæces. The temperature was normal, and remained so throughout the patient's stay in hospital. There were no circulatory or digestive disturbances.

A few days later, Mr. Jones removed the bullet without difficulty from the site above indicated. It proved to be a conical revolver-bullet, about $\frac{3}{4}$ in. long and flattened at the apex from contact with some hard substance. The wound healed readily, and gave no trouble.

On May 19th were noted involuntary twitchings of the legs, which were very constantly present and continued during her stay in hospital. On the 25th, she was found to have some slight power of movement of the fingers of her left hand, and increasing power in the movements of the feet. The faradic current was regularly applied to the limb muscles, the electric reactions of which were normal. Shortly after this she commenced to move the abdominal muscles, and to a slight extent the intercostals: the power of all of these slowly increasing. Towards the middle of June she became able to move slightly the fingers of the right hand, but there was little improvement elsewhere. She complained a good deal of occasional wandering sensations, of "pins and needles," and of the almost constant muscular tremors. From this time, until she went home on July 30th, there was no change, except a slight increase in the power of the already-acquired movements.

By kind permission of Dr. Martin, her medical adviser, I saw this patient again, at her own home, in September. As regards motor power, there was little or no change from the time of her leaving the Infirmary. The limbs were not more wasted than would be expected from her long confinement to bed, and the muscles everywhere presented normal electric reactions. The knee-jerk was much exaggerated, and there was ankle-clonus on both sides; the radial, ulnar, and triceps reactions were absent. She had complete control over her bladder and rectum. There was no anæsthesia. The pupils were normal. She complained greatly of pain in the various joints, but especially in the left elbow and ankle. The general health had remained good.

This case presents so many points of similarity to Case VII. that we are tempted to seek in the latter the explanation of the symptoms. There is the same sudden paralysis with anæsthesia, followed by spastic symptoms, the anæsthesia passing off gradually, the bladder and rectum, at first paralysed, recovering their functions. There is, however, this important difference, that, whereas in Case VII. we have evidence of destruction of a portion of the grey matter of the cord, causing atrophic paralysis and some permanent impairment of sensation, there are here no symptoms but such as might arise from compression of the cord. Further, in the present instance, the symptoms are absolutely symmetrical. We are thus led to look for some cause of general compression of the cord, at a point near the origin of the 4th or 5th cervical nerve. In Case VII., the compressing agent was a central hæmorrhage, causing destruction in its immediate vicinity. In the present instance we can hardly assume this to have been the lesion, as, if we do so, we must suppose an effusion of blood large enough to cause continued pressure on the whole of the structures of the cord, and yet so small as not to have extended into the anterior cornua. If, however, the hæmorrhage were meningeal, lying entirely outside the cord, it might very well cause such pressure without producing any destruction of its substance. Now, a moment's consideration will show, that the accident was of just such a nature as to produce this lesion. An examination of the neck, or of such a plate as that given on p. 302, vol. i., of Quain's 'Anatomy' (8th edition), will show, that if a bullet were to enter "immediately below and behind the left mastoid process," and pursue a straight course to a point "some 3 inches to the right of the 5th cervical spinous process," it would cross the vertebral column about the level of the 4th cervical spine, that is, about the level of the 5th cervical nerve, the upper limit of the paralysis and anæsthesia in our case. Now, the bullet was flattened at the apex as if it had struck some bone, and as it entered below the mastoid process, the only bone in its track would be a portion of the spine. It would then appear that the bullet struck the spinal column somewhere immediately above the 5th cervical nerve, and it probably did not penetrate the cord, or there would not have been the subsequent partial recovery. It appears to us, then, more than probable that it injured in its passage the spinal meninges, giving rise to hæmorrhage sufficiently profuse to compress the cord. It may be said that the instantaneous appearance of the symptoms negatives this view of their production, but we must remember that the only authority for this sudden onset is the patient herself, who was doubtless not at the time in a judicial frame of mind, having been shot by her husband, and having then to witness the latter commit suicide.

That she should fall is not surprising; she would then be confused, probably faint for some little time, and that time would be quite sufficient for the effusion of a quantity of blood from the rachidian veins. It is also possible that a small piece of bone may have been detached from one of the vertebræ and driven in upon the cord, so as to press upon without lacerating it which would naturally cause instant symptoms of compression, but which accords less satisfactorily than does the theory of meningeal hæmorrhage with the partial recovery which ensued.

In concluding a review of the above cases, there are a few points to which reference may be made, and for the consideration of which it is more convenient to regard the cases collectively.

We are at once struck by the relatively large number of cases of hæmorrhage within the vertebral canal, constituting at least three, and probably four, out of a total of nine cases of injury to the cervical region of the spine. The number of these cases occurring in actual practice is probably much under-estimated, many of them being regarded as instances of concussion of the spine, without definite organic lesion. The symptoms are frequently complicated, and without a careful consideration of the origin and course of the affected tracts, would appear to be inexplicable on the hypothesis of a single lesion. If, however, these symptoms be fully investigated with the assistance of an accurate knowledge of the anatomy of the cord, they will probably in most cases be found to be clearly due to some gross local lesion. It is to be regretted that of the numerous reported cases of spinal injury, a minority only are sufficiently detailed to permit of an accurate diagnosis, it being only too customary to use such loose expressions as "partial paralysis of the upper limbs."

Another cause of the comparative neglect of hæmatomyelia as a result of injuries to the spine, is the relative infrequency of post-mortem examinations verifying this condition. These cases are not nearly so fatal as fractures and dislocations, and their true pathology is therefore not fully cleared up. Thus we find that of our five cases of fracture all were fatal, whereas of the four cases of hæmorrhage only one died. Under these circumstances the only possible means of diagnosis is the demonstration that all the symptoms may be due to a single focus of injury; but where such demonstration is practicable, we submit that a diagnosis of hæmorrhage should always be preferred to one which regards the case as an instance of that vague and somewhat theoretical affection, "concussion of the spinal cord."

We notice also the tendency of hæmorrhage into the cord to occur not very far from the level of the 4th or 5th cervical nerve, and we think that a simple mechanical consideration will explain this localisation. In Case IX. the direction taken by the bullet was, of course, a pure accident, and we may therefore omit that case from consideration. But in all the other three instances (Cases VI., VII., and VIII.) of hæmorrhage, we have seen reason to believe that the cord lesion was the result of an acute bend of the cervical portion of the spinal column. Now, a glance at a properly-articulated skeleton or well-drawn picture of the spine will show that the most anterior of the cervical vertebræ are the 4th and 5th, with their intervening disc. If, now, this region of the spine be over-extended, either from a blow on the back of the neck, or from forcing back of the head or trunk, these two vertebræ will form the apex of the angle which is produced, and here, therefore, will be the most acute bend of the cord. Thus it happens that in all such injuries the cord is more stretched at this point than at any other, and is therefore more subject to a laceration of its blood-vessels, and the production of a hæmorrhage.

Finally we may note the relative frequency in our cases of certain well-recognised symptoms of injury to the spine.

As regards *external deformity*, we find this present only in one case (Case I.), there being in none of the others the slightest local indication of the severe injury which had been sustained.

Hyperæsthesia was observed in one case only (Case IX.), but in all the three cases which recovered (Cases VII., VIII., and IX.), there was observed a sensation of *tingling* in the parts previously anæsthetic, as the anæsthesia was gradually passing away.

Priapism was present in all the cases of fracture during the remainder of life. Of the three cases of hæmorrhage occurring in males, it was present in one alone (Case VI.), and even here it existed for about twenty-four hours only. It would thus appear that the fibres connecting the lumbar centre with the brain are situated at some considerable distance from the centre of the cord, so as either altogether to escape, or readily to recover from the pressure of a central hæmorrhage.

In the same way we find that the fibres connected with the bladder-centre are not liable to suffer from prolonged compression by a central hæmorrhage. Thus there was permanent *retention of urine* in all the cases of fracture. On the other hand, looking at the cases of hæmorrhage, we find retention lasting one day only in Case VI., and four days in Case VII.; while in Case VIII. there was no bladder trouble at all. In Case IX., a female, there was incontinence for some weeks, but not permanently. Can it be that the implicated fibres are situated near the periphery of the cord, that they thus escape compression or rapidly recover in the cases of central hæmorrhage; but that, owing to the hæmorrhage in Case IX. being meningeal, they are more directly exposed to a pressure, and therefore incapacitated for a longer period?

The present cases throw no light on the chemical changes which the urine may undergo. Two of them were examined at an early period for sugar and albumin, with negative results. In Case I. decomposition and alkalinity set in about the end of the first week, but the other cases give no indication of the period at which this change usually occurs, all having either died or regained control over the bladder before the fourth day, with the exception of one which was only seen long after the injury.

Bed-sores were noted in Case I. at the time of admission, four days after the accident; all the other cases of fracture died within a few hours, before their development was possible, and they did not appear in any of the cases of hæmorrhage.

The condition of the *pulse* was noted in Cases I., III., V., and VII. In the first three of these it had the characters usually described in lesions of the cervical cord, being slow, full and of low tension. In Case VII. it was quick, but the observations were only made after the setting in of the febrile reaction due probably to myelitis. In Case IX. it was quite normal at the time the patient came under observation, five weeks after the accident. Case III. is, however, evidence that the pulse-rate may increase with rise of temperature, a pulse of 130 being rapid even with a temperature of 103°.

As is usually the case in spinal injuries, the *temperatures* observed were sufficiently remarkable, appearing to bear no relation to the nature and site of the injury ; it is unnecessary to repeat the figures given above, no classification of cases being as yet possible.

The condition of the *pupils and palpebral fissures* is interesting, not only from a scientific point of view, but also because changes are very constant and are highly unlikely to be simulated in any case of attempted deception. Whether or not we accept the existence of a cilio-spinal centre in the cord, there can be no doubt that dilator fibres for the iris, and fibres supplying the muscle of Müller pass downwards to about the level of the last cervical or first dorsal nerve-root, and thence outwards to join the sympathetic by means of the *rami communicantes*, and that these fibres are liable to be injured by any lesion of the cord above this level. The affection may, however, be overlooked if bilateral, being distinctly visible only in a dim light, because in bright light contraction is normally present and therefore would not be remarkable. In Case I. we find imperfect dilatation of the pupil on both sides, lasting until the closing stages, when there was dilatation, owing probably to the iris assuming the position of rest from paralysis of all its muscles, preceding death. In Case II. the pupils were said to be "normal," but this was probably owing to their not being properly examined in diffused light. In Cases III., V., and VI. there was no doubt about the contraction both of the pupils and of the palpebral fissures. In Case IV. the pupils were dilated, but death occurred before the subsidence of the first shock of the accident. In Case VI. the contraction was much more marked on the right side, coincidently with the greater extension of the hæmorrhagic focus on that side. In Case VII., where the lesion was more extensive on the left side, the left pupil only was contracted.

In Cases VIII. and IX., where the injuries were less severe the pupils had altogether escaped. In Case III. the paralysis of Müller's muscle gave rise to a very remarkable sensation of softness and compressibility of the eyeball.

The last symptom to which I wish to refer is the condition of the cutaneous reflexes and tendon reactions below the seat of the lesion. On this subject we have notes in six of the cases only. We see at once that there is an important distinction to be drawn between the condition of the reflexes in the earlier and in the later periods after the injury. As regards *cutaneous reflexes*, they were noted as absent immediately or very shortly after the accident in Cases I., V., and VI., and they were lost later on in Cases VII. and IX.; beyond this there was no examination, but the results are exactly in accordance with the common observation that cutaneous reflexes are usually diminished or lost in cases where communication is cut off between the cerebral cortex and the periphery. The reason of this change is perhaps not yet satisfactorily explained, but as it is generally recognized as a matter of daily observation in all diseases of the nervous system, we need not dwell upon it longer at present.

As regards the *tendon reactions*, we find a most interesting condition. In Cases I., V., and VI. they were absent immediately after the occurrence of the injury, their early condition not being noted in any of the other cases. On *à priori* grounds we would expect that a complete transverse lesion of the cord, high up, would, by removing the influence of cerebral inhibition, tend to increase the activity of the tendon reactions—the knee-jerk, and ankle-clonus. Such is the change soon after a transverse myelitis, after compression of the cord, and after degeneration of the lateral columns. Here, however, the exactly opposite condition is produced. Probably the true explanation of this peculiarity is, that the shock of the accident acts as a powerful depressant of reflex action, and is thus more than able to counterbalance the increased activity which the reflex centres should normally acquire on being relieved from the repressing influence of cerebral inhibition. The facts would thus appear to be of the same order as the well-known observations of Goltz, that upon section of the spinal cord of the dog in the neck the lumbar reflex centres for the bladder, rectum, and generative organs are temporarily paralysed. This condition of paralysis of reflex action from shock is constantly seen clinically in cases of retention of urine following upon operations or injuries of quite distant parts—as, for instance, in a case which I have seen quite recently, where retention of urine lasted for two days after the performance of Mac Ewen's operation for genu valgum. If such be the true explanation of this loss of the deep reflexes, we should expect them soon to be restored to their normal condition, but from the very nature of the cases this is a change which can seldom be observed. Thus Cases V. and VI. died within a few hours of the accident, there being no opportunity for the reflex centres of the cord to recover their functions. Case I., it is true, lingered longer, but he remained throughout in a condition of depressed vitality not favourable to energetic reflex action, and we know well that in many cases of general depression the reflexes are diminished or absent. Further, it is to be remembered that the determination of a slight knee-jerk is by no means easy in a patient necessarily recumbent on a water-bed. But, turning to some of the later cases, we find evidence that the knee-jerk does, as a rule, return before very long, the best example being Case IX., where at the fifth week after the accident the knee-jerk was apparently normal in degree. Finally, we find that in older cases, where there has been time for the development of a descending degeneration of the pyramidal tracts, there is, as is usual under such circumstances, a well-marked intensification of the tendon reactions, as evidenced by Cases VII., VIII., and IX.

SEQUEL TO A "CASE OF OPHTHALMOPLEGIA, COMPLICATED WITH VARIOUS OTHER AFFECTIONS OF THE NERVOUS SYSTEM."[1]

BY J. S. BRISTOWE, M.D., F.R.S.

In my paper I gave reasons for believing the symptoms in this remarkable case to be due to functional disease of the nervous system; and remarked, "A practical advantage in regarding the cases I have cited as functional is, that it fortifies me in the hope, so long as the survivor" (Gertrude H.) "lives, that she may yet recover."

On reference to my paper, it will be seen that the patient had undergone no improvement when she passed from under my care on the 2nd of February, 1885. At my request, she was then admitted into St. Bartholomew's Hospital, where she remained for several weeks without material, if any, change. I had authentic information of her condition not long after she left that institution, at which time also no improvement had taken place. Shortly afterwards, the family removed from the East of London to Walthamstow, and I lost sight of her. But latterly rumours have reached me, through some of the hospital nurses, between whom and her family there has been kept up an irregular correspondence, that the girl had recovered; and on the 16th of November she came to St. Thomas's with her mother to pay me a visit.

She was thinner than she had been, and pale, but otherwise looked quite well. There was no paralysis of the eyes, which worked freely in all directions and consensually; the pupils were equal, and acted to light and accommodation. I thought the tongue, when protruded, tended slightly to the right, but the deviation was scarcely perceptible. She used both arms and both hands with equal facility; and said that her right hand was as useful as her left, but its grip was certainly not so powerful as that of its fellow. She walked perfectly well; but the knee-jerks were perhaps over-brisk. There was no anæsthesia. She had quite lost her headache; and never now complained of sickness or giddiness. It appears that she had had only two fits after leaving St. Bartholomew's; the first, six weeks after returning home, the second, about two months subsequently. I could not ascertain the order of events in her progress towards recovery; but her recovery seems to have been gradual; and she has been well and able to work like any other healthy young woman for the last twelve months.

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[1] Case of Gertrude H. 'Brain,' October 1885, p. 328.

Clinical Cases.

A CASE OF HYSTERO-EPILEPTOID ATTACKS, WITH CONVULSIONS PRODUCED BY LOCAL IRRITATION OF THE SKIN.

BY G. PARKER, M.D.,

Late Resident Medical Officer to the Children's Hospital, Paddington.

In the following case[1] there were two points of interest. 1. A convulsive seizure could be brought on by irritating a spot on the chest, but only if a sufficient time had elapsed since the last attack. If the epileptogenous zone were touched within about an hour from the previous seizure, no effect took place; but after an interval sufficient to permit a certain accumulation of energy, as it were, the patient was at once struck down if touched on a spot between the right nipple and axilla. 2. Very frequent seizures of a hystero-epileptic character occurring in a child, whose family history pointed rather to true epilepsy than to a syphilitic taint, yielded at once to mercury and potassium iodide, after numerous other remedies had been tried to relieve them without success.

Amy R., a healthy-looking child of 11, presenting no signs of premature development, was admitted as an out-patient on March 8. The mother stated that she had of late been subject to convulsive seizures, falling down with a scream at all hours of the day and night. The family history showed that the father's father had been subject to fits, during one of which he was drowned by falling into a river. The father denied any specific taint. Patient is the fourth in a family of nine children. The eldest is now 17, six are alive and healthy, none have had any fits. The mother had no miscarriages. Patient was always quite healthy till three years ago, when early one morning she became suddenly insensible and convulsed. Similar attacks occurred at intervals during six months. No exciting cause was known, but the mother says she noticed that two upper canine teeth were cut at this time.

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[1] This case was under the care of Dr. Laycock, by whose kindness I am allowed to publish the observation.

One month ago, without any discoverable cause, the fits began again. On an average one every hour has occurred during the night, nine during the day. These numbers have remained approximately the same. She generally falls down with a scream, if standing or sitting, and has cut herself in doing so, but has never bitten her tongue or passed her water during a fit. She lies convulsed for one or two minutes; the left arm is flexed and drawn across the chest, the hand grasping forcibly the right side; the trunk is bent forwards to the right, and the head brought down on the chest; the right leg is kicked to and fro spasmodically, especially in those fits which occur during sleep. The movements, in fact, resemble in an exaggerated form those which would be made by a child when tickled in the right axilla and trying to save itself. The mother has noticed that if she is touched at a spot on the thorax between the right nipple and the axilla, an immediate attack comes on, provided that none have occurred within an hour or two previously. This was tested in the consulting room, and a fit induced. She is said to have frequently retained consciousness through her fits.

March 25.—Admitted as in-patient, not having improved under treatment. *Present condition.*—She appears to be well-nourished and cheerful; the general intelligence and memory are very good; bowels regular, appetite fair; nothing abnormal noted in the respiratory or circulatory systems; no signs of approaching catamenia; says that she has sometimes just before a fit a creeping sensation passing up towards the right axilla.

March 26.—She had five fits while in the hospital during the afternoon of yesterday, and six more in the night, each one lasting some seconds only. They commence with a groan, the head falls forward, the left arm is flexed across the chest and the hand grasps the right flank, the mouth and lower part of face are drawn into a sort of grin. To-day a fit was induced by touching the chest in the region of the right nipple. The conjunctivæ were quite insensible during some of the attacks. Seventeen fits occurred in the 24 hours, each lasting about 60 or 90 seconds. She is being carefully watched to discover the existence of masturbation, or any allied cause for the attacks.

March 27.—Seven fits during the day. She is apparently quite insensible to pain during the attacks if tested by a pin. There is no cyanosis or stoppage of respiration.

March 31.—There is tenderness in the region of the left ovary. The left pupil is distinctly larger than the right, she has apparently red blindness in left eye, and the hearing is only half as acute with the left ear as with the right. There is no albumen in the water.

April 2.—Pot. bromide up to gr. xx. in the dose; am. and sodium bromide, belladonna, ergot, oil and steel wine, and valerian are being tried in succession. A slight improvement has followed the use of two or three of these remedies, but they all fail to act after the first few days. The fits to-day are fewer, but stronger, and followed by much exhaustion, which has not been the case previously.

April 5.—One fit last night continued for about five minutes, and was accompanied by foaming at the mouth. The numbers of the attacks from the first inst. have been for each 24 hours 16, 19, 20, 21, 19. The bowels are rather confined, and the temperature is nearly a degree above normal.

April 15.—The daily chart records from 10 to 20 fits every 24 hours. There is full power over the sphincters, knee-jerks taken after a fit were normal; no ankle-clonus is present. No impaired intelligence or aphasia, and no loss of sensation have been made out. Between the attacks she eats and plays like a child in perfect health. When one comes on, she falls suddenly forward with a scream, remains rigid and huddled together for about a minute, has generally some convulsive movements of the arms and legs, chiefly on the right side; the left arm is often dragged over as before. The right side of the face twitches and the eyes are turned to the right. Afterwards she appears to wake up with a smile, and smooths her clothes mechanically, or if it is night, she turns over and falls asleep again quietly. On examination by the ophthalmoscope the discs were seen to be normal, the choroid was thinned at the periphery, and a few spots of pigment appeared through it. R. $\frac{6}{12}$ m.; 5 D. $\frac{6}{6}$ pi. Left, $\frac{6}{18}$ n.; 1 D. $\frac{6}{6}$ pi.

May 4.—The fits are much fewer in the day-time; at night she groans and mutters during the short time they last. The limbs become rigid at the commencement of an attack, and are then flexed. At times there is much exhaustion. In other respects there is no change. Nitrite of sodium is now being tried.

May 13.—Faintness and cyanosis having been produced, the nitrite was stopped finally to-day. The attacks are again more frequent yesterday and to-day.

May 15.—To-day 25 fits are registered in the 24 hours.

May 16.—To-day 8 fits only.

May 24.—The faradic current was applied sharply to spine and limbs.

June 1.—Frequent short applications of the battery, both by night and day, after fits have been tried. The fits, which have for some time been getting fewer in the day-time, now cease to occur except at night, but the number in the 24 hours remains the same. Urethan, valerian, the bromides, and camphor monobromide have all been tried without success.

June 14.—From 6 to 28 attacks occur nightly. To-day the liquor hyd. perchlor. ʒss, potassium iodide gr. iii, and decoction of cinchona were given for the first time.

June 18.—The attacks have fallen to one in the 24 hours.

June 19.—No fits at all to-day.

June 30.—No more attacks; patient a little weak, but otherwise quite well; ordered to become an out-patient, and to continue her medicine.

July 20.—Remains perfectly well. Mother states that she has cut four molar teeth this year.

Sept. 6.—Has had no recurrence of the fits, and remains in perfect health.

The disappearance of the attacks when treated with mercury may of course have been only a coincidence, but the suddenness of the change rather points to a causal relation, possibly by the reduction of some enlarged gland. The induction of the fits by touching a special area resembles the effects produced in certain guinea-pigs under Dr. B.-Séquard. In them, after the division of the sciatic or other large nerve epileptic seizures occurred when the skin of the cheek was irritated; and similar cases have occurred in human beings.[1] It was, however, obviously undesirable to encourage these tendencies in the patient, but sufficient trials were made to establish the fact clearly, as well as the existence of a period in which irritation had no effect. Besides the problems suggested by this period of stable equilibrium, the child's statement, that "a creeping feeling" occurred before some of the fits in the region where a touch would often bring them on, is important with regard to the nature of an aura. Either what is subjectively known as an aura is purely central in origin, and if so, we must say that there was no analogy between it and the touches in this case, though both were equally followed by fits; or it is some local impression, such as the friction of the clothes, which, occurring when the central irritation is at its height, is perceived abnormally, and is the determining cause of a fit, just as much as the touches were.

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[1] See, too, a paper by Dr. Arthur Gamgee in the 'Brit. Med. Journal,' vol. ii. 1878, on Hystero-Epilepsy, and the recent experiments by Mr. V. Horsley discussed before the Pathological Society, Nov. 1886. Is there not an analogy between the state of the central nervous system thus produced, and the tremor which appears peripherally for some time after section of a trunk nerve?

A CASE ILLUSTRATING THE DIFFERENTIAL DIAGNOSIS OF TUMOUR OF THE CORD AND TUMOUR OF THE CAUDA EQUINA.

BY JAMES OLIVER, M.B., F.R.S. (EDIN.).

The cord itself, as such, ceases to exist beyond the level of the lower part of the body of the first lumbar vertebra; whilst the cauda equina forming its continuation, is made up of the roots of the lumbar and sacral nerves as they descend to their respective foramina of exit. The symptoms complained of, and physical signs manifested by, a patient, the subject of a neoplasm invading either the spinal cord or cauda equina, necessarily vary according to the level at which the nerve-tract becomes compressed and so disturbed. The existence, however, of a difference in the structural arrangement of the nerve-fibres in the cord proper and its continuation, the cauda equina, produces a marked variation in the evolution and manifestation of symptoms, resulting from the presence of a new growth invading these two totally distinct parts of the spinal tract.

The bundles of nerve-fibres forming the cauda equina are like those composing any large peripheral nerve-trunk, both motor and sensory in character, which, continuing throughout their course perfectly distinct, end in their respective peripheral expansions, motor or sensorial, free anastomosis apparently of the corresponding terminal filaments being effected.

In the cord proper, however, the motor and sensory tracts do not long continue in apposition, but follow a different course in this structure; decussation, more or less complete, in the case of both, occurring, proximally with the motor, and distally with the sensory, in respect to the higher centre. The motor tract coming from one or other hemisphere of the brain, decussates with its fellow of the opposite side somewhere in the medulla oblongata, and is afterwards transmitted without any further appreciable decussation down one half of the cord, to supply the muscles of that same side of the body. The sensory bundles, on the other hand, resulting from a union of fibres coming from peripheral expansions, enter the cord at varying levels, according to the part of the body in which they take origin, pass almost immediately to the opposite half of the spinal cord, and are thence transmitted to the brain, without further recrossing, in that part of the cord away from the side of the body in which they originate. The motor tracts, as they descend in the cord, give off nerve-fibres to the same side of the body, whilst the sensory, as they ascend, accumulate from the opposite half of the body. If, therefore, one half of the spinal cord be divided completely, the motor fibres supplying that same side of the body below this level are severed from the higher centre, whilst the sensory fibres corresponding with sensorial expansions of the opposite side of the body are interrupted in their course. When a patient suffers from slow compression of the cord—the result of invasion by a neoplasm—the clinical facts occasionally illustrate beautifully this physiological dogma, as in a case which recently came under my care, the leading points of which I shall here relate.

Henry I. ——, æt. 40, two years ago began to complain of pain in the back, extending round the left side, and following apparently the course of the eleventh and twelfth dorsal nerves. Soon afterwards he experienced sensory disturbances—pins and needles—in the right foot. This peripheral sensation gradually extended up the leg, and in the course of a few months he remarked that there was total loss of feeling in the right leg throughout. He was still able to walk about well. Gradually, however, he noticed a tendency to drag the left foot, and in the course of six months he had lost completely all power over the left leg. As yet he had experienced no motor impairment of the right lower limb, nor sensory disturbance in the left. One month later, he noticed a tendency to drag the right, and simultaneously, that he had lost sensation in the left; he had never, at any time, however, experienced any abnormal sensations in the left leg, as in the case of the right. The loss of volitional power over the right leg gradually progressed, and the patient now, two years after the initial manifestation of symptoms, is completely paraplegic, with a total loss of sensation in both legs.

The urine has to be drawn off.

No wasting of muscles of the limbs, which react sluggishly to the faradic and constant currents.

Skin plantar-reflex increased.

Knee-jerk well marked—somewhat augmented.

Ankle-clonus can be developed, although feebly.

In the case of tumour invading the cauda equina pain may be occasionally, although not invariably, complained of. There is noted gradual loss of power in the lower limbs, beginning at first in one leg, and eventually extending to the other.

Loss of volitional power is accompanied by a gradual, or sometimes rapid wasting of muscle tissue. These muscles, over which the patient has little or no volitional control, fail to respond to the faradic or constant current, whilst those which continue to respond to the volition of the patient, although somewhat enfeebled, will still react to electric excitation, faradic or galvanic. Usually there is some defect in the tactile, and painful sensibility; as, however, sensation may apparently be carried on by a free nerve-anastomosis, this may not be a marked symptom of the disorder. When there is evidence of sensory disturbance, it is experienced first, and, as a rule, much more markedly in the limb which first shows signs of motor enfeeblement; and not, as in the case of tumour invading the cord, primarily in the limb opposite to that which reveals initially the signs of motor impairment.

The bladder in such cases, as in the case of tumour invading the lumbar enlargement of the cord, has its functions disturbed, the patient being as a rule unable to empty this viscus of its contents.

There is absence of skin plantar-reflex, no ankle-clonus, and invariably it is impossible to elicit any knee-jerk.

Abstracts of British and Foreign Journals.

Philosophische Studien. Herausgegeben von WILHELM WUNDT. Dritter Band; pp. 691. Leipzig: Wilhelm Engelmann, 1886.—Prof. Wundt's 'Archiv' contains much that is of interest to the readers of 'BRAIN,' and must not be ignored on the supposition that it is given over to metaphysical discussion and commentaries on Kant. The editor's name is sufficient indication of the scientific point of view adopted, and he expressly states at the close of the first volume that the chief aim of the 'Studien' is the building up of experimental psychology. In looking over the three volumes so far issued[1] it will be seen that about half of the papers are accounts of experimental work done by Professor Wundt's pupils in the psychological laboratory at Leipzig, while about half of the remainder discuss methods of experimental research. Valuable results in psycho-physics had been obtained by individual workers, or published from physiological and physical laboratories. But Professor Wundt took a step of great importance in founding in 1879 a special laboratory for psycho-physical research. A similar laboratory was started in 1883 at Johns Hopkins University, and others are about to be formed at Cambridge and the University of Pennsylvania; but the most systematic and valuable work done during the past seven years in psycho-physics comes from Professor Wundt's laboratory, and is described in the 'Philosophische Studien.' It would lead too far to give even a brief summary of this work, but it may be worth the while to indicate its scope so that those interested can refer to the original papers. About half of the experiments described are directed towards determining the time taken up by mental operations, a direction of research to which the term 'psychometry' might be confined. The reaction time has been measured under varying conditions, as also the time needed in perceiving, willing and in the association of ideas. On the work with which the names of Weber and Fechner are associated, that is the relation between the strength of the stimulus and the intensity of the sensation (psycho-physics in its narrower sense), experiments on light and sound have been published and others are now being carried on. The analysis of sensation, to which we may reckon contrast, has proved itself to be one of the most fertile fields of investigation, including as it does the researches of Helmholtz on sound, and the many valuable experiments on the retina and sense of sight. The extent and limits of consciousness is a subject of considerable interest and open to experimental study. Investigations have been made at Leipzig, and are still being prosecuted on the time-sense, under which we may include the estimation and comparison of intervals and the time relations in which sensations stand. Lastly, the conditions of memory, on which Ebbinghaus has made interesting experiments, may be mentioned. These six branches of research are those which have been taken up at Leipzig, and do not of necessity cover the whole field of psycho-physics, which is itself but one branch of psychology.

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[1] Numbers are published as material is accumulated; during the past five years twelve such numbers have been issued, making up the three volumes.

Experimental psychology enjoys the advantage of having its way to some extent cleared by physics and biology, and can borrow from these sciences apparatus and methods; but any one working in psycho-physics will find that he must himself devise and control the greater part of the apparatus needed, not finding ready, for example, such a purely physical and elementary fact as the time it takes an electro-magnet to attract its armature. Still more must the experimenter rely on himself in arranging methods; these depend to a large extent on the very consciousness we wish to investigate, and their advantages and shortcomings can often only be seen after the experiments have been made. I mention these facts, to explain why the work described in the 'Studien' is not all of equal value; when similar experiments are continued by the same or different workers, improvements in method and apparatus are constantly being made, so that the later results are the more accurate and valuable.

I give below a classified list of the papers which describe experimental work. In some cases a paper covers more ground than the heading under which it is entered.

J. McKeen Cattell, Ph.D.

PSYCHOMETRY.

'Ueber die Apperceptionsdauer bei einfachen und zusammengesetzten Vorstellungen.' Max Friedrich. i. 39–78.

'Experimentelle Untersuchungen über die Association der Vorstellungen.' Martin Trautscholdt. i. 213–251.

'Ueber die Einwirkung einiger medicamentöser Stoffe auf die Dauer einfacher psychischer Vorgänge.' Emil Kraepelin. i. 417–463 and 573–606.

'Ueber die Unterscheidung von Schallstärken.' Ernst Tischer. i. 495–543.

'Ueber die einfache Reactionszeit einer Geruchsempfindung.' W. Moldenhauer. i. 606–615.

'Die zeitlichen Verhältnisse der Willensthätigkeit.' Julius Merkel. ii. 73–128.

'Ueber die Zeit der Erkennung und Benennung von Schriftzeichen, Bildern und Farben.' James McKeen Cattell. ii. 635–651.

'Ueber den Einfluss der Reizstärke auf die Dauer einfacher psychischer Vorgänge mit besonderer Rücksicht auf Lichtreize.' G. O. Berger. iii. 38–94.

'Psychometrische Untersuchungen.' James McKeen Cattell. iii. 264–305 and 452–493.

PSYCHO-PHYSICS.

'Zur Frage der Gültigkeit des Weber'schen Gesetzes bei Lichtempfindungen.' Emil Kraepelin. ii. 306–327.

'Die Methode der richtigen und falschen Fälle in ihrer Anwendungen auf Schallempfindungen.' Gustav Lorenz. ii. 394–475.

ANALYSIS OF SENSATIONS.

'Untersuchungen über den Farbencontrast vermittelst rotirender Scheiben.' Bruno Schmerler. i. 279–305.

'Bemerkungen über die Messung von Schallstärken, mit Rücksicht auf psychophysische Versuche.' Ernst Tischer. i. 543–556.

'Die Messung von Schallstärken.' Paul Starke. iii. 264–305.

'Ueber die Anwendung der Methode der mittleren Abstufungen auf den Lichtsinn.' Alfr. Lehmann. iii. 497–534.

LIMITS OF CONSCIOUSNESS.

'Untersuchungen über den Umfang des Bewusstseins bei regelmässig auf einander folgenden Schalleindrücken.' Georg Dietze. ii. 362–394.

'Ueber die Trägheit der Netzhaut und des Sehcentrums.' James McKeen Cattell. iii. 94–128.

TIME-SENSE.

'Untersuchungen über den Zeitsinn.' Julius Kollert. i. 78–90.

'Neue Versuche über den Zeitsinn.' Volkmar Estel. ii. 37–66.

'Zur Lehre vom Zeitsinn.' Max Mehner. ii. 546–603.

'Ueber die Zeitverhältnisse der Apperception einfacher und zusammengesetzter Vorstellungen, untersucht mit Hülfe der Complicationsmethode.' Woldemar von Tchisch. ii. 603-635.

MEMORY.

'Untersuchungen über das Tongedachniss.' H. K. Wolfe. iii. 534–572.

Abstracts of Papers on Nervous Diseases published in St. Thomas's Hospital Reports from 1875 to 1884 (vols. vi. to xiv. inclusive). By W. B. Hadden, M.D. (Lond.)

1875.

"Some Cases of Sudden Recovery from Mental Disease." By W. Rhys Williams, M.D.

The recovery in each case was permanent, and in this respect differed from many of the recorded cases in which recovery was transient.

1876.

"Notes on Cases of Nervous Disorder." By W. M. Ord, M.B , F.R.C.P.

The author first discusses reflex irritation of the skin, and the part played by reflex irritation in producing certain morbid conditions of the skin. Four cases are cited in which eczema appeared to arise from irritation, mechanical and catarrhal, of the urinary organs and passages. Acne results from irritation of the mucous membrane of the genital passages, and the psoriasis and prurigo which are common at the climacteric period, are probably related with local changes. There are reflex relations between eczema and bronchial catarrh, these conditions sometimes alternating in the same individual. Patchy baldness of the scalp is often associated with anal pruritus. The reflex influence exerted by the skin on internal organs is illustrated by several cases in the author's practice. He concludes that it is possible to rouse "reflex trophic influence in wasting limbs by stimulation of a dormant central ganglion through the peripheric nerves."

"Intra-Cranial Aneurysms." By Thomas B. Peacock, M.D., F.R.C.P.

This paper contains an admirable *résumé* of this subject up to date. No less than eighty-six cases are summarised, and the conclusions to be drawn from them are given in great detail. The paper is well worth consulting for reference.

1877.

"Statistical Report of Cases of Chorea." By Thomas B. Peacock, M.D., F.R.C.P.

This is an elaborate analysis of ninety-two cases of chorea occurring in the author's practice. It is well adapted for reference, and would prove an admirable guide for future observers.

"On Simple Meningitis." By W. S. Greenfield, M.D. Lond.

Two groups of cases are embraced in this paper: (1) those that are truly primary; (2) those arising as a complication or sequela of acute disease, or as the result of some blood contamination. In the first class are included sporadic cases of cerebro-spinal meningitis, and the form which occurs in infancy without known cause; in the second class, which is mainly discussed and illustrated, are grouped the cases depending on such conditions as pneumonia, rheumatism, pyæmia, septicæmia, syphilis and Bright's disease. The paper contains some excellent and suggestive observations on the pathology of this affection.

1878.

"Cases of Chorea." By J. S. Bristowe, M.D., F.R.C.P.

The author remarks that he has never seen a post-mortem examination in chorea without heart disease having been detected, although its existence is not always recognised during life. In one of the cases given there was complete loss of speech, and the choreic movements were most marked on the right side. Attention is drawn to cases in which paralysis (paraplegia or hemiplegia) may precede or replace the chorea. Impairment of common sensation, general or unilateral, is sometimes observed, resembling closely that found in hysteria. Absolute insanity supervenes in rare instances. In two of the cases recorded, epileptic attacks existed, and in one of these insanity came on. In the other, the choreic symptoms were preceded, and in part, accompanied by a succession of epileptic fits. Occasionally chorea becomes chronic, and in two such cases seen by the author the lower limbs became paralysed and stiff, suggesting degenerative change in the lateral columns of the cord.

1879.

"On Hysteria and Hystero-Epilepsy." By William H. Stone, M.B., F.R.C.P. "Also Further Remarks on Hystero-Epilepsy."

The author discusses the term "hysteria" at some length, showing how its meaning has become modified, owing "to the tendency to metaphor and metathesis which is the besetting sin of medical terminology." A classification of functional neuroses is suggested, each group being illustrated by cases. The author narrates his experience of *le transfert* as seen in Charcot's wards at Salpêtrière, and then proceeds to point out what he considers weak points in the demonstration. In the second paper the author gives two typical cases presenting the symptoms described by Charcot, which came under his notice since the first paper was written. In one of these cases the anaesthesia disappeared when a magnet was placed close to the affected limb. Elaborate precautions were taken to exclude the effects of expectant attention.

1881.

"On the Use of the Continued Current in Diabetes." By William H. Stowe, F.R.C.P., and Walter J. Kilner, M.B.

In the two cases recorded an ascending current from the nape of the neck to the forehead was employed, the negative pole being placed in the former region, the positive in the latter. Some temporary improvement seemed to result from this treatment; but in the first case a large carbuncle formed at the back of the neck where the negative pole had been applied, and the patient died.

"Contribution to the Pathology of Double Optic Neuritis." By Walter Edmunds, M.D.

This paper is based on a case of head injury which proved fatal in twenty-four hours. There was a fracture of the middle fossa of the base of the skull, but it did not pass into the orbit or into the immediate neighbourhood of the optic nerves. These latter exhibited well-marked early inflammatory changes. There was some basal meningitis, and the author believes that the simplest explanation of the appearance described "is that the inflammation spread from the inflamed meninges to the tissue between the nerve sheaths."

"Nerve-stretching in a Case of Spinal Meningitis with Ataxic Symptoms, due to Injury." By W. H. Armstrong.

The author claims that this was one of the first cases of nerve-stretching done in this country, and the result was more successful, than that of most cases hitherto published. By the operation the patient recovered cutaneous sensibility, the lancinating pains were much relieved, and the power of locomotion was greatly improved. In this case the left sciatic nerve was stretched, and the author is inclined to believe that an effect is produced on the nerve centre, not on the periphery.

"On Cases of Injury to the Optic Nerve." By Edward Nettleship.

The author points out that the optic nerve seldom undergoes repair and regains its function after injury. This depends in great measure on the usually severe nature of the injury, possibly also on the anatomical peculiarities of the optic nerve. When the nerve is divided in front of the optic foramen, the movements of the eyeball may interfere with the apposition of the cut ends. The paper is illustrated by a series of valuable cases.

1882.

"On Paraplegic Rigidity in Hemiplegia."

By W. B. Hadden, M.D.

The author calls attention to the fact, that a lesion in one cerebral hemisphere gives rise to direct as well as crossed paralysis; and that in late hemiplegic rigidity the lower limb of the opposite side may become the seat of ankle-clonus, and occasionally even becomes contractured like its fellow. It is also observed that in bilaterally associated nerve-nuclei, irritation on one side, as in the case of inhibition, reacts on the opposite side. Moreover, bilaterally associated nerve cells are less apt to undergo atrophic changes than cells not bilaterally associated. It seems likely that paraplegic rigidity in hemiplegia depends on the variations in the decussation of the pyramidal-tract fibres in the medulla, and not on a secondary decussation of the motor fibres in the spinal cord. It is possible that the fibres of the direct pyramidal tract enter into relation with the multipolar cells in the lower part of the cord.

"Some Effects of Brain Disturbance on the Hand-writing."

By W. H. Stowe, F.R.C.P.

This paper contains some interesting observations on agraphia, illustrated by the author's personal experience during an obscure illness.

"Clinical Remarks on so-called Painful Paraplegia."

By J. S. Bristowe, M.D., F.R.S.

The paraplegia to which attention is called depended in all the five recorded cases on malignant growths, involving the spinal cord or the nerves proceeding from it. In two instances the lumbar vertebræ were affected, and in the remaining three the growth originated in the periosteum of the ilium.

"Paralysis of Serratus Magnus." By H. H. Clutton.

Among the causes of the paralysis the author gives:—typhoid fever, progressive muscular atrophy, blows and strains, and possibly infantile paralysis. In three cases observed by him no definite cause could be assigned. The anatomical and clinical aspects of the paralysis are fully discussed, and the views of Erb, Duchenne, Bäumler, Vivian Poore, and others, compared and criticised.

"On the Electrical Resistance of the Human Body." By W. H. Stowe, F.R.C.P.

This is an abstract of a paper read before the British Association at Southport. The author believes that the electrical resistance of the human body is enormously overstated. The obstacles to its accurate determination and the means adopted to overcome them are stated in detail. The paper will be of interest to experts.

1883.

"Two Cases of General Atrophy treated by the 'Weir Mitchell' Method." By Seymour J. Sharkey, M.B.

The author's object in writing this paper is to record his success in following out the treatment advocated by Dr. Playfair. Both cases resulted in cure. An excellent account of the method of treatment and the class of cases to which it is applicable is given. The author observes that "too great prominence is given to the nervous system. In the published cases prostration of the nervous system has not been more prominent than prostration of the muscular, glandular, and every other system."

"Cases of Tubercular Meningitis." By John S. Bristowe.

All the cases present particular features of interest, or are specially instructive. (1.) In the first three cases the meningeal affection was obviously secondary, as is usually the case; but the interesting point is that the primary tubercular disease had become retrogressive, and, clinically speaking, a cure had been effected. (2.) In the next three cases the growth of tubercles on the membranes of the brain was apparently slow, and symptoms were developed only when inflammation was superadded, or the growth became so abundant as to involve the surface of the brain itself or the cranial nerves. (3.) Tuberculosis of the membranes, with or without meningitis, is apt to supervene in cases of tubercular tumours of the brain. (4.) In one case the tubercular meningitis was primary, no trace of tubercular disease being found elsewhere in the body. (5.) In one case the symptoms closely resembled delirium tremens. (6.) In conclusion, the author analyses some of the main symptoms occurring in his cases, *e.g.* distension of the ventricles, optic neuritis, paralysis of the cerebral nerves, hemiplegia, convulsions, and temperature.

"Cases of Tubercle of the Cerebellum." By J. S. Bristowe.

The most important facts are :—(1) the existence of the common signs of intracranial tumours, namely, headache, sickness and optic neuritis; (2) the headache was by no means limited to the back of the head; (3) the almost complete absence of anything like a staggering gait, excepting in the last case, in which it had been of long duration and was typical; (4) the supervention in the last case of tubercular meningitis.

"A Case of Spasmodic Wryneck treated by Excision of a portion of the Spinal Accessory Nerve." By Charles A. Ballance.

The patient was a married woman, aged 48, who had suffered severely from spasmodic torticollis for ten years. During the previous ten months the affection had become very severe, and interfered seriously with the taking of nourishment and with sleep. Drugs, electricity, and massage had been tried without avail. The muscles affected were the right sterno-mastoid and the upper portion of the right trapezius. The nerve supplying them, the external branch of the right spinal accessory, was exposed, and about one inch excised, the proximal end at the same time being stretched. The spasm disappeared at once.

Five months later the right sterno-mastoid and trapezius were found to be atrophied. There was some tendency to muscular spasm when she was excited or when the face was directed upwards, but she could by an effort prevent any movement of the head. It was probable that the deep origin of the posterior primary division of the left sub-occipital nerve and also the trapezial fibres of the left spinal accessory were exposed to the same irritation as the deep origin of the right spinal accessory.

The portion of nerve excised was found healthy on microscopical examination.

Mr. Ballance gives an account of the literature of the subject, and also discusses the surgical means to be employed in such cases.

"Clinical Observations on Chorea." By W. B. Hadden, M.D.

The author discusses some of the more unusual symptoms of chorea under five heads, illustrating each group of cases. (1.) Paralysis may be the predominant symptom, preceding the choreic movements, and persisting long after their disappearance. The paralysis often attacks parts which have been little affected by the movements. The paralysis to which attention is called is identical with that occurring in hysteria. In one of the recorded cases the patellar tendon reflexes were absent. (2.) Complete loss of articulation is illustrated by three cases, in all of which the choreic movements were chiefly right-sided. Speechlessness, like the motor paralysis in chorea, is transitory, both probably depending on inhibition of motor centres, and not necessarily due to exhaustion by antecedent spasm. The seat of the lesion may be in Broca's convolution, or in the co-ordinating centre in the medulla oblongata. (3.) Anæsthesia is not very uncommon in chorea. There seems to be no rule as regards its distribution; although in most of the cases seen by the author the anæsthesia, when unilateral, was limited to the side chiefly affected by the movements, or, when bilateral, was more marked on that side. (4.) Hysteroid convulsive attacks are probably rare, although emotional disturbance is frequent. In the case given the chorea was ushered in by the convulsive attack.

It is pointed out that symptoms of a hysterical nature often supervene in chorea. The author illustrates the converse, the supervention of genuine chorea in hysteria. (5.) Chorea in pregnancy. In most cases there is a previous history of chorea or rheumatism, so that pregnancy can only be looked upon as a possible exciting agent. In one case the tendency to recurrence in succeeding pregnancies is illustrated.

"Epilepsy." By John Harley, M.D.

This paper is the outcome of 300 cases of epilepsy observed by the author. Many of his conclusions are corroborative of previous experience, but some are not usually recognised as belonging to so-called simple or idiopathic epilepsy. The following are some of the facts which appear to result from this paper. Epilepsy is essentially a disease of early life. The period of adolescence is more prone to epilepsy than any other. The proneness is greater in females than in males.

During the prime of life (from 20 to 50) the liability is less than half that of the adolescent period. After 50, the liability still more rapidly declines, but in the case of males shows a tendency to rise after the age of 60. Chronic epilepsy is more common in men. The attacks in males are more frequently interrupted by long quiescent intervals, these intervals being much longer in males than in females. Epilepsy may result from imitation or fright. Consciousness may be preserved, partially or completely, during the fit. Consciousness may be suspended, without convulsion. Evanescent insanity, as the immediate consequence of the fit, is common. Disorders of sensation may precede, accompany, or follow the epileptic attack. Disorders of motion may occur, as well as partial paralysis of the tongue and loss of speech. Frequent repetition or violence of the attacks will sometimes produce complete exhaustion of the nervous system and death. (In one of the two cases given a small cysticercus was found in the brain, but there was no sign of irritation or inflammation in the neighbourhood.) Hemiplegia is a frequent result of epilepsy, and even infantile paralysis may result from it.

Onufrowicz on the Auditory Nerve.—Onufrowiez's paper (*Archiv f. Psych.*, xvi. p. 711) is based upon an examination of the brains of two rabbits, whose internal ears had been destroyed on one side as completely as possible by an operation through the outer ear. One rabbit lived six months, the other two and a half months. The brains were subjected to a microscopic examination, and the resulting atrophies noted. The posterior root of the auditory nerve was much atrophied on one side, the anterior root was affected in a considerably less degree. The following are the conclusions of the author:—(1) The so-called "external auditory nucleus," or, as he prefers to call it, Deiters' nucleus, has nothing whatever to do with the auditory nerve. The nuclei were equal in size and healthy on both sides. (2) It is more than doubtful whether the so-called "internal auditory nucleus" has any direct connection with the auditory nerve. (3) The auditory tubercle (Stieda's tuberculum laterale, Stilling's Nacken des Kleinhirnschenkels) is the nucleus of origin of the posterior root of the auditory nerve, that is, of the auditory nerve proper. The auditory fibres of the auditory nerve are in all probability contained only in its posterior root, the anterior root containing the fibres that are distributed to the ampullæ of the semicircular canals. The auditory tubercle is a mass of grey matter that lies to the outer side of the posterior root, and is sometimes described as a portion of the flocculus. It is the homologue of the superior corpora quadrigemina, the centre for the optic nerves, and of the posterior cornua of the cord, the centre for the sensory nerves. The fibres of the posterior auditory root enter the tubercle only after passing through the anterior auditory nucleus, which is the homologue of the spinal ganglia. (4) The place of origin of the anterior root of the auditory nerve is either the vermiform process, or the grey substance of the 4th ventricle underlying the superior cerebellar peduncles, or in both. (5) The striæ medullares or acusticæ are not direct continuations of the auditory nerve; they are either secondary fibres that issue from the auditory tubercle, or they have, as Longet held, no connection at all with the auditory nerve. (6) Of the further central connections of the auditory nerve we are entirely ignorant. The paper contains a valuable sketch of the views of previous investigators as to the origin of the auditory nerve, and the author emphasizes the superiority of the experimental method of anatomical investigation over even the most careful purely histological researches.

Richter on the Optic Radiations.—Richter (*Archiv f. Psych.* xvi. p. 639) reports the case of a man who, for more than a year, had suffered from homonymous left hemianopsia and other nervous symptoms. The post-mortem revealed on the right side adhesion of the pia mater to the cortex of the three occipital convolutions, the cuneus and the posterior half of the occipito-temporal convolutions, median and lateral; and there was disorganisation of the cortex in these places. There were less marked changes on the inferior surface of the occipito-temporal lobe as far as the tip of the temporal lobe. The white matter underlying the cortex in these regions exhibited fat granule cells, and the tract of degeneration was followed through the centrum ovale along the outer wall of the lateral ventricle into the pulvinar of the optic thalamus. The corpora quadrigemina, corpora geniculata and optic tracts and nerves were quite intact. The author criticises Monakow's statements that occipital lesions are followed by descending degeneration of the anterior corpora quadrigemina, corpora geniculata externa and optic tracts and nerves, and affirms that in man, at all events, occipital lesions have only been proved to cause secondary degeneration of the pulvinar. Another tract of degeneration was traced from the tip of the temporal lobe through the tegmentum to the crusta.

Atrophy of both optic nerves, or even of one, is followed by reduction in the ganglion cells of both optic thalami, all four corpora quadrigemina and all four corpora geniculata, but the author has not observed any change in the occipital cortex.

Westphal on a Peculiar Group of Symptoms.—The patient (*Archiv f. Psych.* xvi. pp. 496 and 778) was a man aged 47, free from syphilitic or alcoholic taint. The first symptoms were paresis of the internal rectus of the left eye, slight double ptosis, vertigo, and other diffuse cerebral symptoms. Subsequently the lower extremities became weak, and the weakness developed into almost complete paralysis; the upper extremities were affected similarly, but not to such a degree. In the lower extremities various groups of muscles began to display slight degrees of rigidity, the patellar reflexes became heightened, and at a later stage the paradoxical contraction was elicited by movements of the ankle, knee, and hip joints. As the disease progressed, the weakness of the upper extremities increased to absolute paralysis in the case of certain movements; muscular rigidity showed itself, and also the paradoxical contraction. At a later period the muscles of the jaw were affected. The facial muscles showed no marked abnormality.

The motor symptoms were not the only symptoms present, there were striking sensory disorders. Loss of sensibility was first observed in the peripheral portions of the upper and lower extremities, and in the short space of about two years the anæsthesia had extended over almost the entire surface of the trunk and extremities, and ultimately the region of the fifth nerve was affected. The muscular sense was lost, but it is a noteworthy fact that sensibility to heat and cold was always retained, that the superficial reflexes were still evocable, and that the muscles continued to react to electrical stimulation and did not display the usual atrophic degenerative changes. During the whole course of the case, there were no special cerebral symptoms, with the exception of disturbed sleep, vertigo, and feelings of nervous apprehension, and the intelligence of the patient was unimpaired till within a short time of his death. The patient died after an illness of about three years.

The diagnosis of the case was doubtful. It resembled a case of multiple cerebro-spinal sclerosis in some respects—but the widespread anæsthesia, and the low degree of muscular rigidity in the extremities, negatived this view. The post-mortem disclosed disease of the posterior columns of the cord, atrophy of the sensory and motor spinal nerves, and a peculiar change in the tibialis anticus muscle, which was the only one examined; there were a few scattered patches of disease in the lateral columns.

The disease of the posterior columns extended along their whole length, but the root-zones were unaffected, and this probably accounts for the fact that the patellar reflex was retained. The absence of ataxia, notwithstanding the extensive disease of the posterior columns, is attributed to the slight intensity of the disease, and to death occurring before its full development; it may also perhaps be due to the peculiar localisation of the disease, for we do not yet know what portion of the posterior columns have to be diseased to produce ataxia. The sensory symptoms are explained by the affection of the peripheral nerves, which can be considered either as a primary atrophy or as a chronic parenchymatous neuritis, and which probably had its origin in the distal terminations of the nerves. The peculiar changes in the muscles, Westphal thinks, may be related to the paradoxical contraction.

Rumpf on Syphilis, Infantile Paralysis, and Cerebellar Atrophy.—*Syphilis of Nervous System.*—A man, aged 31 (*Archiv f. Psych.* xvi. p. 410), contracted syphilis when 29, and subsequently suffered from a cutaneous eruption and sore throat, and from a suddenly developed attack of right hemiplegia of the limbs. Symptoms of spastic paralysis of the left leg next showed themselves, then incontinence of urine and fæces. The patellar reflexes were exaggerated on both sides, a slight girdle sensation was present, and a few months before death sensibility became impaired in both lower limbs. The patient died at the age of 32. The chief post-mortem results were:—(1) An old lesion in the left internal capsule, with secondary degeneration of the pyramidal tracts of the right lateral column of the cord; (2) degeneration of the left lateral column, chiefly of its pyramidal tracts, in the cervical region; (3) degeneration of Goll's columns in the cervical regions; (4) a patch of inflammation in the dorsal portion of the cord, implicating the lateral and posterior columns of both sides, and gradually increasing in dimensions till the lumbar region was approached, where the disease again contracts its limits, and is confined to the posterior columns, and to the postero-lateral parts of the lateral columns; (5) the blood-vessels throughout the cord, but especially in its dorsal region, presented proliferative changes in their three coats, the inner coat being affected later than the outer coats—these changes are often found associated with syphilis, but they are not pathognomonic of it, being found in other diseases.

Infantile Paralysis.—A case of infantile paralysis (*polio-myelitis anterior acuta*) is described in which there was atrophy of the central convolutions of the cerebrum. The paralysis affected the right side, and came on in the fourth year. Death occurred from pneumophthisis in the eighteenth year. The middle and lower parts of the central convolutions of the left cerebral hemisphere were smaller than the corresponding parts of the right. The author states that the dimensions of the left internal capsule (in front of the genu) and of the left lenticular nucleus were markedly less than those of the corresponding structures on the right side. He regards the cerebral atrophy as an arrested development, the result of the spinal paralysis.

Cerebellar Atrophy.—The paper closes with the case of a cat which had symptoms of cerebellar disease, and which was found after death to have atrophy of the cerebellum.

Schulz on Aphasia, Hemianopsia, Spinal Sarcoma, and Tetanus.—Schulz (*Archiv f. Psych.* xvi. p. 579) reports the following four cases:—

Aphasia.—A man aged 20 suddenly fell from his cart on March 21 without losing consciousness. On March 24 his speech became affected, and occasional convulsive movements were noticed in his right face. On March 29 he was admitted into Hospital suffering from complete amnesic aphasia, nevertheless he understood all that was said to him, and could repeat words that were uttered slowly to him. There was paresis of the lower half of the right face, the tongue deviated to the right, and every three or four minutes there were strong convulsive twitchings in the whole of the right face, with temporary loss of consciousness. The aphasia became absolute, the convulsions continued to grow worse, and ultimately became general, and the patient passed into a comatose condition and died on March 31. The autopsy disclosed a cheesy tumour, that was probably tubercular and was of the size of a hazel nut, in the inferior frontal convolution of the left hemisphere (Broca's convolution).

Hemianopsia.—A woman aged 49 had temporal hemianopsia of the left eye (18 months before, she had suddenly felt something come over her left eye), a reeling gait, occasional vertigo, and right hemiparesis; and her mind was childish. Under iodide of potassium treatment she improved, but in two months it was noticed that there was complete left homonymous hemianopsia, the right eye being now affected. Six months afterwards, on April 1, 1884, the patient was admitted into hospital much worse; the hemianopsia remained; there was paresis of all four extremities, which gradually developed into almost complete paraplegia, with contracture of the limbs, heightened tendon reflex and sensory disturbances. At a later stage there was paralysis of the sphincters and extensive bedsores, and the patient died on Sept. 3, 1884.

During the last three months she was demented. The diagnosis was :—Tumour springing from the hypophysis cerebri compressing the right optic tract, and latterly implicating the pons. The autopsy, however, showed chronic cerebral pachy- and leptomeningitis, interstitial encephalitis, and hypertrophic cervical pachymeningitis at the level of the 3rd and 4th cervical vertebræ. The pia mater was especially thick and adherent in the region of the chiasma, and to this Schulz is inclined to attribute the hemianopsia, though he admits the evidence is not at all conclusive. The spinal cord was softened opposite the 3rd and 4th cervical vertebræ.

Spinal Sarcoma.—A girl of sixteen had complained for three months of pain in the back, and during the last three weeks had been troubled with numb feelings in the hands and arms, and her arms had gradually become paralysed. On admission into hospital on March 31, 1881, there was almost complete paralysis of both upper and lower limbs; loss of sensibility over the whole of the upper limbs, and vaso-motor disturbances evidenced by hot, red, and swollen hands; diminished sensibility in the lower limbs, with partial loss of the deep and superficial reflexes, and paresis of the bladder. Acute pains were still felt in the back, and soon respiration became affected, and the patient died on April 2, 1881. The diagnosis was acute meningo-myelitis. At the post-mortem a tumour was found ensheathing the cord from the cauda equina upwards to the upper cervical region. It had the characters of an alveolar sarcoma, and had evidently originated in the pia mater. In some places, notably in the upper dorsal part of the cord, and the upper cervical part, what little of the cord was left, was completely softened.

Tetanus.—The author reports a mild case of tetanus in which the irritability of the facial nerves to mechanical stimulation was increased, but their irritability to the stimuli of the constant current was normal. This last observation agrees with Erb's statements, but it is in opposition to what other authorities have found in this disease.

W. J. Dodds, M.D., D.Sc.

Report of a slight Outbreak of Epidemic Cerebro-spinal Meningitis, with Notes of Six Cases. By Wm. Frew, M.B., C.M.—This outbreak occurred in the town of Galston, near Kilmarnock. It originated, the author thinks, through the bad sanitary conditions of the place; there was an accumulation of the sewage in the neighbourhood of the houses. Personal intercourse, however, could be traced from the first patient down to the last (with one *possible* exception).

Case I.—Initial symptoms, headache and vomiting: on second day, temperature 104°, followed by delirium, sordes on lips, &c. By fourth day, improvement set in. No rash was observed. After recovery, absolute deafness, staggering gait. Girl, age 10.

Case II.—Æt. 1. Vomiting and pain. Temperature 104°. Measly rash on forearms and limbs. Death within fourteen hours of onset.

Case III.—Sister of Case II. Age 7 years. Rigor, headache, abdominal pain. Next day, vomiting. Third day, temperature 103°–104°, much restlessness and pain in head. Fourth day, worse; photophobia, feeling of constriction at throat, mottled purplish rash on forearms and legs; legs flexed on abdomen, retraction of head; delirium, &c., towards night. Sixth day, improvement, tenderness along spine. Recovery tedious, relapses of pain, vomiting, &c., much exhaustion; but no paralysis or affection of special senses.

Case IV.—Girl, $8\frac{1}{2}$ years old. A very acute case; chief symptom, vomiting; death in a single night. *Post-mortem*, petechial spots on legs: hyperæmia of membranes, with inflammatory exudation beneath them, at vortex of brain, over cerebellum, and along spinal cord.

This girl had visited Case III. three days before her own seizure.

Case V.—Vomiting, fever, convulsions affecting principally the left arm and left angle of mouth. No rash. Death on fourth day.

Post-mortem.—Calvaria of a bright pinkish colour: venous congestion of surface of brain; gelatino-purulent exudation on upper and under surface of brain (especially right anterior part), and also over cerebellum and spinal cord.

Case VI.—Age 14 months. Vomiting, fever, convulsions, mouth and eyes drawn to right. Examination of the fundus oculi showed no neuritis, but distinct hyperæmia. No rash observed. Death on fourth day.

On the Pathology of Pseudo-hypertrophic Muscular Paralysis, with Remarks on a so-called Degeneration of the Nervous System. By G. S. Middleton, M.A., M.D.—Two cases were examined—in the first case the microscopic changes were as follows:—

Muscular system.—The muscular fibres varied greatly in thickness; some muscles (and especially those which were otherwise least abnormal) were in a condition of hyaline degeneration; in others there was increase of connective tissue between the fibres, or infiltration with fat. In the heart and bladder there was increase of interstitial connective tissue, but no fatty infiltration, nor hyaline degeneration.

Nervous system.—The only change found was the presence of small whitish glancing bodies, scattered in large numbers, and indifferently (though more numerous in the white substance than in the grey) through all parts of the nervous system that were examined. They stained in logwood, and faintly with osmic acid. These the author justly concludes to have been produced by the method of preparation, the parts having been hardened in alcohol.

In Case II. the muscular tissue presented a marked fatty infiltration, some increase of connective tissue, and atrophy of muscular fibre. No hyaline degeneration. Nervous system practically normal.

From a consideration of these cases, and a review of the literature of the subject, the author concludes that the disease is essentially a myopathy.

Beevor on Three Cases illustrating the Localisation of Motor Centres in the Brachial Enlargement of the Spinal Cord (*Roy. Med. Chir. Soc.*, April 14, 1885).—These cases illustrated the groups of muscles found by Ferrier and Yeo (*Proc. Roy. Soc.* No. 212, 1881), to be thrown into action when each motor root in the monkey's brachial plexus was faradized. In the first case—progressive muscular atrophy—in the fourth cervical group the deltoid, rhomboid, biceps, brachialis anticus, supinator longus, extensors of fingers were severely affected on both sides, and in addition on the left the supra- and infra-spinati; the fifth cervical (clavicular part of deltoid, biceps, brachial. antic., supin. long. serrat. mag., extensors of fingers) was completely paralysed; in the sixth cervical (lat. dorsi, pect. maj. triceps, serrat. mag., pronators of wrist), only the two latter, and the last only slightly; in the seventh cervical (lat. dorsi, subscap., pect. maj., triceps, teres major, flexors of fingers), the teres was affected on both sides, and the pect. maj. slightly on the left and the flexors of the fingers on the right; in the eighth cervical (long flexors of wrist, ulnar flexors of wrist, small muscles of hand, extensors of fingers, triceps (long head), pect. maj.), all the muscles were severely affected on the right, except the last two, and extensors of fingers and pectoralis maj. slightly paralysed on the left; the first dorsal (small muscles of hand and interossei), on right only. The above shows the biceps type, viz. fourth and fifth cervical on both the right and left, besides the eighth cervical and first dorsal on the right. No reaction to faradic current was obtained in the severely affected muscles.

The two other cases—triceps type—occurred in boys of 18 and 11 years old, who had infantile paralysis when young.

In one case on the right side, the only muscles affected were the triceps. latissimus dorsi, pectoralis major (sternal part only); these were absolutely powerless, and the last muscle had completely disappeared; they gave no reaction to faradic or constant currents; they belong to the sixth and seventh cervical groups. On the left side, beside these muscles, all muscles corresponding to the seventh cervical, except flexor digit., and all those of the sixth cervical, except pronators of wrist, were paralysed; in the fifth cervical group, the deltoid, serrat. mag., extensors of wrist were only slightly affected; and in the fourth cervical, only the rhomboid, supra- and infra-spinati, teres minor were severely affected: there was no reaction to the constant current in the severely paralysed muscles.

In the other triceps case, the teres major, latiss. dorsi, subscap., pect. major (lower half), triceps, serratus magnus and the rhomboid (?), were the only muscles paralysed, and these, excepting the two last, are supplied entirely by the sixth and seventh cervical roots, so that the muscles corresponding to the biceps type have escaped.

It should be mentioned that Ferrier has since found (*Proc. Roy. Soc.*, No. 225, 1883) that his original list, used in this paper, is one root too high, and the groups of muscles should refer from the fifth cervical to the second dorsal, instead of from the fourth cervical to the first dorsal, so that the group here assigned to the fourth belongs to the fifth, &c.

Case of Tubercular Tumour of the Cerebellum. By WILLIAM VOST, M.B., C.M. (*Glasgow Medical Journal*, July 1884.)—Onset of symptoms ten days before death; purpuric rash, cough malaise; then staggering gait: pain left side. On admission, could not walk, lay with knees flexed, pain on extending them. Pupils large, not responding to light; eyes fixed, but slight to and fro movements of them later on. Tâche cérébrale; bladder partly paralysed. Semi-comatose condition. Temperature 100°–102·4°. *Post-mortem*, phthisis, with old pleurisy of left lung; and a tumour (tubercular) on the under-surface of the left lateral hemisphere of the cerebellum.

J. A. Ormerod, M.D.

Remak on a Case of Bilateral Paralysis of the Spinal Accessory Nerve (*Deutsche Med. Wochenschrift*, No. 27, 1885. Case exhibited at Verein für innere Medicin, June 1885).—The patient, a man, aged 35 years, contracted indubitable syphilis in 1873. Six months before being exhibited to the Society he began to suffer from pains in the neck and right shoulder, with inability to raise the shoulders; six weeks subsequently his voice became husky.

He presented the following state:—Over the sternal end of the right clavicle an exostosis as large as a hen's egg, and a similar but smaller exostosis over the sternal end of the left clavicle; hard prominences from the right side of the upper cervical vertebræ, and from the left side of the sixth and seventh cervical vertebræ; marked atrophy of the trapezii, levatores anguli scapulæ, rhomboides, muscles of the back of the neck and of the pectorales; the sterno-cleidomastoides only slightly wasted, and the extrinsic muscles of the larynx healthy; both deltoides fairly developed, but the muscles on the flexor aspect of the upper arm atrophied, especially on the left side, on which the supinator longus is similarly affected; muscles on the extensor side of both forearms only rather flaccid; those on the flexor side and muscles of hands intact Motor paralyses exactly corresponding to the distribution and degree of the atrophy easily demonstrated; characteristic separation of the inner border of the scapula from the chest wall when the arm is raised, shows the left serratus magnus to be also involved. Respirations 24–27 per minute, without stridor or dyspnœa, thoracic in type, with respiratory epigastric recession (diaphragmatic paralysis); pulse-rate, 90–115 per minute; acceleration possibly secondary to hurried respiration only; occasional fibrillar twitching of sterno-cleidomastoideus, deltoideus, pectoralis, biceps, and supinator longus on both sides.

On electrical examination, platysmata, muscles of the root of the tongue and extrinsic muscles of the larynx react normally; sterno-mastoides react to moderately powerful faradic current;

but trapezii and left biceps excitable only by very strong current, and their contraction slow and incomplete (faradic degenerative reaction). Galvanic degenerative reaction marked in trapezii, sterno-mastoides, left biceps, and supinator longus; present, but to a less degree, in deltoides; in flexors of arm on right side, and extensors of wrist on left side.

The hoarseness of voice suggested the probability of the intrinsic laryngeal muscles, supplied by the inner division of the spinal accessory nerve, being affected. On April 1st, Dr. Böcker observed the signs of paralysis of the left recurrent laryngeal nerve; on May 6th the right vocal chord appeared the more immobile; on May 16th both chords occupied a position intermediate between the cadaveric and that characteristic of paralysis of the crico-arytænoides postici. On phonation the chords do not approximate, and vibrate coarsely, attributable to paralysis of the crico-thyroides, supplied by the external laryngeal nerve.

There are no signs or symptoms of implication of any of the cranial nerves, except slight difficulty in swallowing, explicable by involvement of the pharyngeal filaments of the spinal accessory (Burchard).

The author considers a syphilitic process—probably a pachymeningitis, extending laterally as far as the ligamentum denticulatum, and involving the anterior roots of the upper cervical nerves, and of the spinal accessory as far down as the fifth and sixth nerves—the most likely explanation of the phenomena observed.

The interest of the case culminates in the laryngeal condition, which the author considers as a fact opposed to the well-known views of Semon regarding the special liability of the abductor fibres to become paralysed in disease affecting the nucleus of the spinal accessory or its peripheral branches. Such an argument appears hardly sound. The chords seem to have occupied a position exactly corresponding to partial abductor paralysis, as has been elsewhere pointed out by B. Fraenkel.

Hughes Bennett on Locomotor Ataxy, without Disease of the Posterior Columns of the Spinal Cord. (Vol. xviii. *Clinical Society's Transactions*, 1885.)—Dr. Bennett gives the details of a case which appears to us to be unique from many points of view. It is in itself not only of great clinical interest, but the conclusions which the author draws from the facts are in our opinion calculated to throw an important light on the physiology and pathology of so-called tabes dorsalis.

A gamekeeper, 48 years old, having previously been in good health, and never having suffered from syphilis or other illness, about nine months before he came under observation gradually experienced attacks of shooting-pains in his legs, and inability to walk properly. This was accompanied with a sensation of numbness in the limbs. All these symptoms slowly increased in severity. On examination, the general health was found good, and all the functions and organs of the body normal, with the following exceptions:

There were occasional slight attacks of giddiness, and slight nystagmus, otherwise the cerebral functions and special senses were normal. The movements of the upper limbs were somewhat weak, and slightly but distinctly ataxic. The bladder, rectum, and sexual organs appeared to be normal. The lower limbs presented all the typical characters of ataxy of an advanced type, the incoördination being markedly increased in the dark, or with the eyes closed. The plantar reflexes were diminished, and the knee-jerk on both sides was totally abolished. In the feet and legs the cutaneous sensibility was slightly diminished and retarded; otherwise and elsewhere it was normal. There were frequent attacks of severe lancinating pains in the lower limbs. The nutrition of the muscles in their electrical reactions was normal. During a residence of two and a-half months in the hospital all these symptoms somewhat rapidly augmented, and the patient died of exhaustion.

On post-mortem examination the essential lesion was found to consist of a series of isolated sarcomatous tumours, situated chiefly in the posterior portion of the pia mater of the cord. These involved the posterior spinal roots, without causing their serious degeneration. These tumours were most numerous in the lower dorsal and lumbar regions. The posterior columns of the cord were everywhere perfectly normal. The medulla oblongata to the naked eye appeared natural, but the microscope revealed a sarcomatous growth occupying the floor of the fourth ventricle, and extending downwards to the centre of the wall surrounding the central canal.

From these facts, of which only a superficial sketch is here given, the author draws important pathological and clinical conclusions. The point to which special attention is directed is this: while during life the patient presented most of the characteristic phenomena of tabes dorsalis, the posterior columns of the cord were found after death to be everywhere normal in structure, while, on the other hand, the posterior spinal roots alone were extensively involved in the morbid lesion. Thus evidence is adduced to show that, for the production of those groups of symptoms comprised under the term of tabes dorsalis, disease of the posterior columns of the cord is not an essential factor, and that they may be fairly developed, the centre being in a perfectly normal condition. The case supports the view, that incoördinate movements may be produced by interruption of the afferent elements of the nervous system. It moreover forms an important pathological link between the classical lesion in the posterior root-zones, and the cases of "nervo-tabes" périphérique of Déjérine.

Here we can do no more than merely hint at the leading points of this valuable observation, and must refer our readers to the original paper for the facts in detail, as well as for a close and scientific reasoning thereon by the author. A series of beautifully executed lithographs of the natural eye and microscopical appearances are appended to the communication.

Hughes Bennett on a Case of Cerebral Tumour, the Surgical Treatment by Rickman J. Godlee. (Vol. lxviii., *Med. Chirurg. Trans.* 1885.)—The incomplete reports of this case which appeared in the different medical journals at the time of its occurrence excited a considerable amount of interest in the profession. In the *Medico-Chirurgical Transactions* for 1885 appears the first authoritative statement from the medical men in charge of the case. This consists of a full and complete record of all the circumstances connected with what we believe to be still a unique case. It will be unnecessary to abstract them in detail, as the general results are now well known. Those interested in the subject will do well to consult the original paper, which is carefully drawn up and fully illustrated. The history and condition on examination of the patient prior to treatment is described, the reasons for the diagnosis discussed, and the necessity for heroic treatment defended. The various steps taken to remove the tumour from the substance of the brain are detailed, and all the phenomena succeeding the operation are recorded. The post-mortem appearances are described, and the condition of the cerebral cortex demonstrated by means of engravings.

Finally, a commentary on all circumstances of the case is given, dealing chiefly with the question of diagnosis, the surgical operation, the clinical phenomena following the operation, and the revelations of the autopsy. In the course of the discussion many interesting facts are demonstrated, and suggestive ideas advanced, on the question of cerebral tumour and the possibility of removing it by surgical operation. The authors conclude by saying that, "Although unfortunately in this instance life was not permanently preserved, the experience we have gained by this case leads us to the belief, that there is an encouraging prospect for the future of cerebral medicine and surgery; and that, as a tumour of the brain can be diagnosed with precision, and successfully removed without immediate danger to life, we confidently anticipate that under more favourable circumstances the operation will be performed with lasting benefit to the patient." Their previsions, as the reader is aware, have been justified by the results recently obtained by Horsley and others; and the surgical treatment of cerebral tumours situated in the motor region has been proved in several cases to be both safe and effectual.

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